WEST Search History

updated search 10/12/05 RP

Hide Items | Restore | Clear | Cancel

DATE: Wednesday, October 12, 2005

Hide?	Set Name	Query	Hit Count
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR	
☐	L1	staphylococcal near accessory near regulator	8
☐	L2	('5587288' |'5976792')!.ABPN1,NRPN,PN,TBAN,WKU.	4
☐	L3	(sar near a) and (sar near r)	8
☐	L4	L3 not l1	8
☐	L5	L3 not l1	8
☐	L6	(sara) and (sarr)	8
☐	L7	L6 not l5	8
☐	L8	L6 not l1	8

END OF SEARCH HISTORY

WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Wednesday, October 12, 2005

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR		
☐	L1	staphylococcal near accessory near regulator	8
☐	L2	('5587288' |'5976792')!.ABPN1,NRPN,PN,TBAN,WKU.	4
☐	L3	(sar near a) and (sar near r)	8
☐	L4	L3 not l1	8
☐	L5	L3 not l1	8
☐	L6	(sara) and (sarr)	8
☐	L7	L6 not l5	8
☐	L8	L6 not l1	8
☐	L9	L6 not l1	8
☐	L10	(sar-a) and (sar-r)	0
☐	L11	(sara) and (sar-r)	0
☐	L12	(sar-r) and staphyloco$	0

END OF SEARCH HISTORY

```
SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1951-2005/Oct 11
         (c) format only 2005 Dialog
  File   5:Biosis Previews(R) 1969-2005/Oct W1
         (c) 2005 BIOSIS
  File  34:SciSearch(R) Cited Ref Sci 1990-2005/Oct W1
         (c) 2005 Inst for Sci Info
  File  35:Dissertation Abs Online 1861-2005/Sep
         (c) 2005 ProQuest Info&Learning
  File  48:SPORTDiscus 1962-2005/Apr
         (c) 2005 Sport Information Resource Centre
  File  65:Inside Conferences 1993-2005/Oct W2
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  File  91:MANTIS(TM) 1880-2005/Jun
         2001 (c) Action Potential
  File  94:JICST-EPlus 1985-2005/Aug W2
         (c)2005 Japan Science and Tech Corp(JST)
  File  98:General Sci Abs/Full-Text 1984-2004/Dec
         (c) 2005 The HW Wilson Co.
  File 135:NewsRx Weekly Reports 1995-2005/Oct W1
         (c) 2005 NewsRx
*File 135: New newsletters are now added. See Help News135 for the
complete list of newsletters.
  File 144:Pascal 1973-2005/Oct W1
         (c) 2005 INIST/CNRS
  File 149:TGG Health&Wellness DB(SM) 1976-2005/Oct W1
         (c) 2005 The Gale Group
  File 156:ToxFile 1965-2005/Oct W2
         (c) format only 2005 Dialog
  File 159:Cancerlit 1975-2002/Oct
         (c) format only 2002 Dialog
*File 159: Cancerlit is no longer updating.
Please see HELP NEWS159.
  File 162:Global Health 1983-2005/Sep
         (c) 2005 CAB International
  File 164:Allied & Complementary Medicine 1984-2005/Oct
         (c) 2005 BLHCIS
  File 172:EMBASE Alert 2005/Oct 12
         (c) 2005 Elsevier Science B.V.
  File 266:FEDRIP 2005/Jun
        Comp & dist by NTIS, Intl Copyright All Rights Res
  File 369:New Scientist 1994-2005/Jul W1
         (c) 2005 Reed Business Information Ltd.
  File 370:Science 1996-1999/Jul W3
         (c) 1999 AAAS
*File 370: This file is closed (no updates). Use File 47 for more current
information.
  File 399:CA SEARCH(R) 1967-2005/UD=14316
         (c) 2005 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
Alert feature enhanced for multiple files, etc. See HELP ALERT.
  File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
         (c) 1998 Inst for Sci Info
  File 444:New England Journal of Med. 1985-2005/Sep W4
         (c) 2005 Mass. Med. Soc.
  File 467:ExtraMED(tm) 2000/Dec
         (c) 2001 Informania Ltd.
```

==========

Welcome to the SIB BLAST Network Service

If results of this search are reported or published, please mention that the computation was performed at the SIB using the BLAST network service. The SIB BLAST network service uses a server developed at SIB and the NCBI BLAST 2 software.

In case of problems, please read the online BLAST help.
If your question is not covered, please contact <helpdesk@expasy.org>.

NCBI BLAST program reference [PMID:9254694]:
Altschul S.F., Madden T.L., Schäffer A.A., Zhang J., Zhang Z., Miller W., Lipman D.J. Gapped BLAST and PSI-BLAST: a new generation of protein database search programs. Nucleic Acids Res. 25:3389-3402(1997).

==========

Query: 115 AA (of which 15% low-complexity regions filtered out)
Date run: 2005-10-12 14:04:30 UTC+0100 on sib-gm1.unil.ch
Program: NCBI BLASTP 1.5.4-Paracel [2003-06-05]
Database: EXPASY/UniProtKB
2,420,647 sequences; 793,847,190 total letters
UniProt Knowledgebase Release 6.2 consists of:
UniProtKB/Swiss-Prot Release 48.2 of 11-Oct-2005: 195589 entries
UniProtKB/TrEMBL Release 31.2 of 11-Oct-2005: 2212675 entries

Taxonomic view | NiceBlast view | Printable view

List of potentially matching sequences

Send selected sequences to Clustal W (multiple alignment) Submit Query
Select up to...

☐ Include query sequence

	Db	AC	Description	Score	E-value
☐	tr	Q6GED9	_STAAR Staphylococcal accessory regulator A homologue [...	190	7e-48
☐	tr	Q6G727	_STAAS Staphylococcal accessory regulator A homologue [...	190	7e-48
☐	tr	Q7A425	_STAAN SarR protein [sarR] [Staphylococcus aureus (stra...	190	7e-48
☐	tr	Q7A2M3	_STAAM Staphylococcal accessory regulator A homolog [sa...	190	7e-48
☐	tr	Q7A064	_STAAW SarR protein [sarR] [Staphylococcus aureus (stra...	190	7e-48
☐	tr	Q5HDR3	_STAAC Staphylococcal accessory regulator R [sarR] [Sta...	190	7e-48
☐	tr	Q9F0R1	_STAAU SarR [sarR] [Staphylococcus aureus]	190	7e-48
☐	tr	Q5HLV6	_STAEQ Staphylococcal accessory regulator R [sarR] [Sta...	158	2e-38
☐	tr	Q8CNC4	_STAEP SarR protein [SE1868] [Staphylococcus epidermidis]	158	3e-38

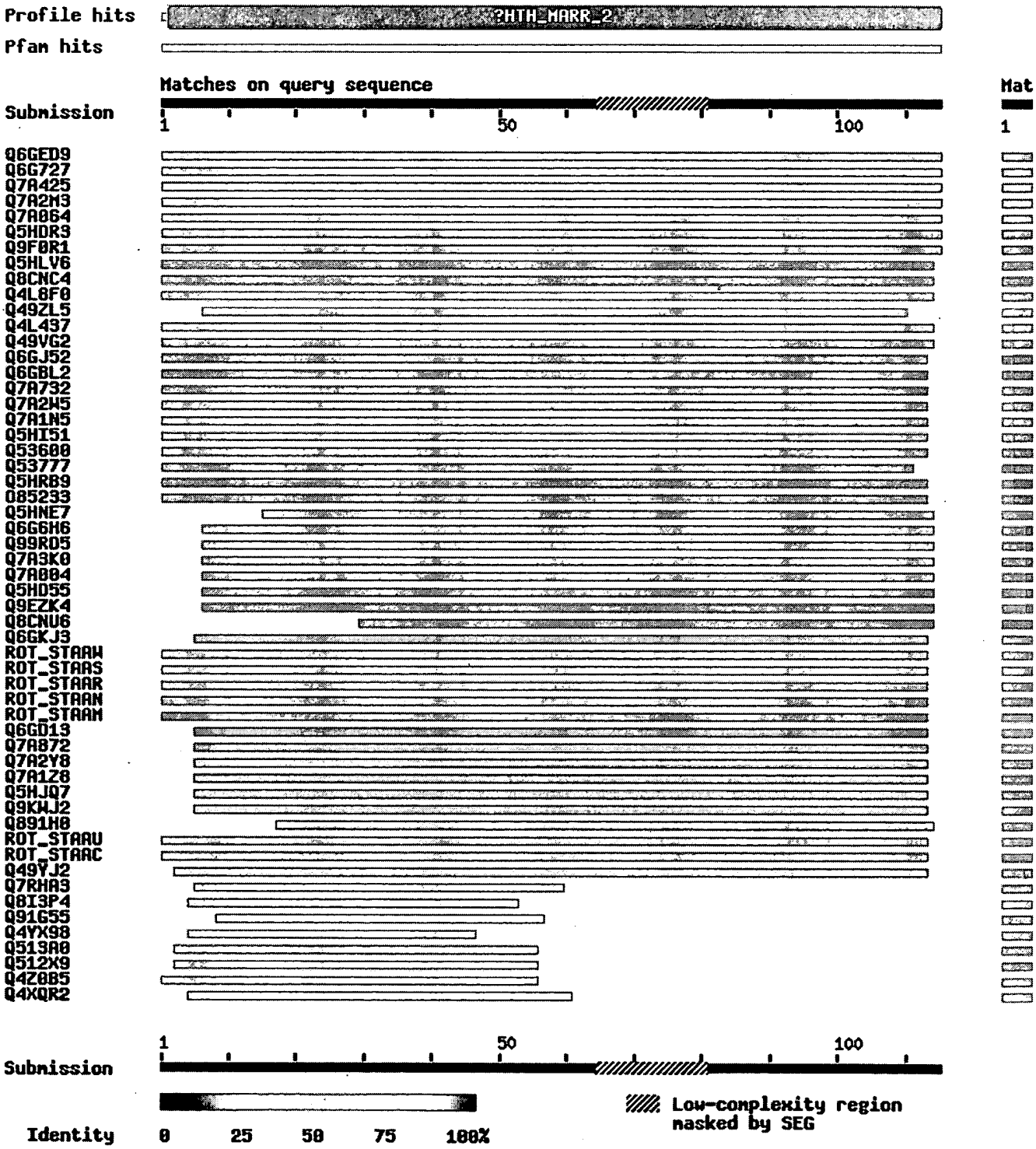
☐	tr Q4L8F0 _STAHJ SarR protein [sarR] [Staphylococcus haemolyticus...	149	2e-35
☐	tr Q49ZL5 _STASA Staphylococcal accessory regulator R [SSP0616] [...	91	5e-18
☐	tr Q4L437 _STAHJ Staphylococcal accessory regulator A [sarA] [Sta...	52	2e-06
☐	tr Q49VG2 _STASA Staphylococcal accessory regulator A [SSP2103] [...	52	2e-06
☐	tr Q6GJ52 _STAAR Staphylococcal accessory regulator A [sarA] [Sta...	49	3e-05
☐	tr Q6GBL2 _STAAS Staphylococcal accessory regulator A [SAS0584] [...	49	3e-05
☐	tr Q7A732 _STAAN Staphylococcal accessory regulator A [sarA] [Sta...	49	3e-05
☐	tr Q7A2W5 _STAAM Staphylococcal accessory regulator A [sarA] [Sta...	49	3e-05
☐	tr Q7A1N5 _STAAW Staphylococcal accessory regulator A [sarA] [Sta...	49	3e-05
☐	tr Q5HI51 _STAAC Staphylococcal accessory regulator A [sarA] [Sta...	49	3e-05
☐	tr Q53600 _STAAU Staphylococcal accessory regulator variant (Stap...	49	3e-05
☐	tr Q53777 _STAAU SarA [sarA] [Staphylococcus aureus]	48	4e-05
☐	tr Q5HRB9 _STAEQ Staphylococcal accessory regulator A [sarA] [Sta...	45	4e-04
☐	tr O85233 _STAEP Staphylococcal accessory regulator A [sarA] [Sta...	45	4e-04
☐	tr Q5HNE7 _STAEQ Repressor of toxins [rot] [Staphylococcus epider...	45	5e-04
☐	tr Q6G6H6 _STAAS Putative staphylococcal accessory regulator [SAS...	41	0.005
☐	tr Q99RD5 _STAAM Staphylococcal accessory regulator A homolog [sa...	41	0.005
☐	tr Q7A3K0 _STAAN SarH2 protein [sarH2] [Staphylococcus aureus (st...	41	0.005
☐	tr Q7A004 _STAAW SarH2 protein [sarH2] [Staphylococcus aureus (st...	41	0.005
☐	tr Q5HD55 _STAAC Staphylococcal accessory regulator U [sarU] [Sta...	41	0.005
☐	tr Q9EZK4 _STAAU Rot-like protein Rlp [rlp] [Staphylococcus aureus]	41	0.005
☐	tr Q8CNU6 _STAEP Repressor of toxins Rot [SE1435] [Staphylococcus...	38	0.060
☐	tr Q6GKJ3 _STAAR Putative regulatory protein [SAR0115] [Staphyloc...	36	0.23
☐	sp Q7A0L8 ROT_STAAW HTH-type transcriptional regulator rot (Repr...	35	0.30
☐	sp Q6G8G5 ROT_STAAS HTH-type transcriptional regulator rot (Repr...	35	0.30
☐	sp Q6GFT9 ROT_STAAR HTH-type transcriptional regulator rot (Repr...	35	0.30
☐	sp Q7A514 ROT_STAAN HTH-type transcriptional regulator rot (Repr...	35	0.30
☐	sp Q99TA4 ROT_STAAM HTH-type transcriptional regulator rot (Repr...	35	0.30
☐	tr Q6GD13 _STAAS Putative regulatory protein [SAS0086] [Staphyloc...	35	0.30
☐	tr Q7A872 _STAAN SarH1 protein [sarH1] [Staphylococcus aureus (st...	35	0.30
☐	tr Q7A2Y8 _STAAM Staphylococcal accessory regulator A homologue [...	35	0.30
☐	tr Q7A1Z8 _STAAW SarH1 protein [sarH1] [Staphylococcus aureus (st...	35	0.30
☐	tr Q5HJQ7 _STAAC Staphylococcal accessory regulator S [sarS] [Sta...	35	0.30
☐	tr Q9KWJ2 _STAAU Hyothetical protein (SarH1) [orfX] [Staphylococc...	35	0.30
☐	tr Q891H0 _CLOTE Hypothetical protein CTC02406 [CTC02406] [Clostr...	35	0.51
☐	sp Q9RFJ6 ROT_STAAU HTH-type transcriptional regulator rot (Repr...	34	0.67
☐	sp Q5HF12 ROT_STAAC HTH-type transcriptional regulator rot (Repr...	34	0.67
☐	tr Q49YJ2 _STASA Repressor of toxins [SSP1002] [Staphylococcus sa...	34	0.67
☐	tr Q7RHA3 _PLAYO Hypothetical protein (Fragment) [PY04086] [Plasm...	33	1.9
☐	tr Q8I3P4 _PLAF7 Hypothetical protein PFE1095w [PFE1095w] [Plasmo...	32	2.5
☐	tr Q91G55 _IRV6 043L [Chilo iridescent virus (CIV) (Insect irides...	31	7.4
☐	tr Q4YX98 _PLABE Hypothetical protein (Fragment) [PB000235.02.0] ...	31	7.4
☐	tr Q513A0 _ENTHI Hypothetical protein [87.t00006] [Entamoeba hist...	30	9.6
☐	tr Q512X9 _ENTHI Hypothetical protein [87.t00028] [Entamoeba hist...	30	9.6
☐	tr Q4Z0B5 _PLABE Hypothetical protein [PB103359.00.0] [Plasmodium...	30	9.6

☐ tr Q4XQR2 _PLACH Hypothetical protein (Fragment) [PC000212.04.0] ... 30 9.6

Graphical overview of the alignments

Click here to resubmit your query after masking regions matching PROSITE profiles or Pfam HMMs

(? Help) (use ScanProsite for more details about PROSITE matches)



Alignments

tr Q6GED9 Q6GED9_STAAR **Staphylococcal accessory regulator A homologue [sarR] [Staphylococcus aureus (strain MRSA252)]** 115 AA align

Score = 190 bits (482), Expect = 7e-48
Identities = 98/115 (85%), Positives = 98/115 (85%)

```
Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF
Sbjct: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
           KPYY                 RSLQDERTVIVYVTDTQKANIQKLISELEEYIKN
Sbjct: 61  KPYYLTKALQKLKDLKLLSKKRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
```

tr Q6G727 Q6G727_STAAS **Staphylococcal accessory regulator A homologue [SAS2185]** 115 AA align

Score = 190 bits (482), Expect = 7e-48
Identities = 98/115 (85%), Positives = 98/115 (85%)

```
Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF
Sbjct: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
           KPYY                 RSLQDERTVIVYVTDTQKANIQKLISELEEYIKN
Sbjct: 61  KPYYLTKALQKLKDLKLLSKKRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
```

tr Q7A425 Q7A425_STAAN **SarR protein [sarR] [Staphylococcus aureus (strain N315)]** 115 AA align

Score = 190 bits (482), Expect = 7e-48
Identities = 98/115 (85%), Positives = 98/115 (85%)

```
Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF
Sbjct: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
           KPYY                 RSLQDERTVIVYVTDTQKANIQKLISELEEYIKN
Sbjct: 61  KPYYLTKALQKLKDLKLLSKKRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
```

tr Q7A2M3 Q7A2M3_STAAM **Staphylococcal accessory regulator A homolog [sarR] [Staphylococcus** 115 AA

aureus (strain Mu50 / ATCC 700699)] align

Score = 190 bits (482), Expect = 7e-48
Identities = 98/115 (85%), Positives = 98/115 (85%)

```
Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF
Sbjct: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
           KPYY                RSLQDERTVIVYVTDTQKANIQKLISELEEYIKN
Sbjct: 61  KPYYLTKALQKLKDLKLLSKKRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
```

tr Q7A064 **SarR protein [sarR] [Staphylococcus aureus (strain MW2)]** 115 AA
Q7A064_STAAW
align

Score = 190 bits (482), Expect = 7e-48
Identities = 98/115 (85%), Positives = 98/115 (85%)

```
Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF
Sbjct: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
           KPYY                RSLQDERTVIVYVTDTQKANIQKLISELEEYIKN
Sbjct: 61  KPYYLTKALQKLKDLKLLSKKRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
```

tr Q5HDR3 **Staphylococcal accessory regulator R [sarR]** 115
Q5HDR3_STAAC **[Staphylococcus aureus** AA
(strain COL)] align

Score = 190 bits (482), Expect = 7e-48
Identities = 98/115 (85%), Positives = 98/115 (85%)

```
Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF
Sbjct: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
           KPYY                RSLQDERTVIVYVTDTQKANIQKLISELEEYIKN
Sbjct: 61  KPYYLTKALQKLKDLKLLSKKRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
```

tr Q9F0R1 **SarR [sarR] [Staphylococcus aureus]** 115 AA
Q9F0R1_STAAU align

Score = 190 bits (482), Expect = 7e-48
Identities = 98/115 (85%), Positives = 98/115 (85%)

```
Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
```

```
          MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF
Sbjct: 1  MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
           KPYY                 RSLQDERTVIVYVTDTQKANIQKLISELEEYIKN
Sbjct: 61  KPYYLTKALQKLKDLKLLSKKRSLQDERTVIVYVTDTQKANIQKLISELEEYIKN 115
```

tr Q5HLV6 Q5HLV6_STAEQ **Staphylococcal accessory regulator R [sarR] [Staphylococcus epidermidis (strain ATCC 35984 / RP62A)]** 114 AA align

```
 Score =  158 bits (400), Expect = 2e-38
 Identities = 80/114 (70%), Positives = 88/114 (77%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           M KI DINDLVNATFQVKKFFRDTKK++NLNYEEIYILNHIL+SESNEISSKEIA CSEF
Sbjct: 1   MGKIKDINDLVNATFQVKKFFRDTKKQYNLNYEEIYILNHILKSESNEISSKEIATCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           KPYY                 RS+ DERTVIV+V+D Q+  I+KLI ELE YIK
Sbjct: 61  KPYYLTKALQKLKDLNLLSKKRSVHDERTVIVFVSDEQREKIEKLILELENYIK 114
```

tr Q8CNC4 Q8CNC4_STAEP **SarR protein [SE1868] [Staphylococcus epidermidis]** 114 AA align

```
 Score =  158 bits (399), Expect = 3e-38
 Identities = 80/114 (70%), Positives = 88/114 (77%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           M KI DINDLVNATFQVKKFFRDTKK++NLNYEEIYILNHIL+SESNEISSKEIA CSEF
Sbjct: 1   MGKIKDINDLVNATFQVKKFFRDTKKQYNLNYEEIYILNHILKSESNEISSKEIATCSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           KPYY                 RS+ DERTVIV+V+D Q+  I+KLI ELE YIK
Sbjct: 61  KPYYLTKALQKLKDLNLLSKKRSVHDERTVIVFVSDEQREKIKKLILELENYIK 114
```

tr Q4L8F0 Q4L8F0_STAHJ **SarR protein [sarR] [Staphylococcus haemolyticus (strain JCSC1435)]** 114 AA align

```
 Score =  149 bits (375), Expect = 2e-35
 Identities = 75/114 (65%), Positives = 87/114 (75%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           M +I DINDLVNATFQVKKFF+DTKKK+NLNYEE+YILN+I RS++NEI+SKEIA  SEF
Sbjct: 1   MGQIKDINDLVNATFQVKKFFKDTKKKYNLNYEEVYILNYIARSKTNEITSKEIATYSEF 60

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           KPYY                 RS+QDER VIVYVTD Q+  I KLI+ELE+YIK
```

```
Sbjct: 61  KPYYLTKALQKLKDLQLLSKKRSVQDERIVIVYVTDEQRDKINKLIAELEKYIK 114
```

tr Q49ZL5 Q49ZL5_STASA **Staphylococcal accessory regulator R [SSP0616] [Staphylococcus saprophyticus subsp. saprophyticus ATCC 15305]** 115 AA align

```
 Score = 91.3 bits (225), Expect = 5e-18
 Identities = 45/104 (43%), Positives = 67/104 (64%)

Query: 7   INDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXX 66
           +N+L++   Q KKFF+ +K+++ LNYEEI+ILN+I   E NEI++K+IAK SE +PYY
Sbjct: 9   LNELISTYQQGKKFFKFSKREYKLNYEEIFILNYIYNCEDNEITAKDIAKHSELQPYYLT 68

Query: 67  XXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELE 110
                        RS  DERTV+VYV + Q+ +I ++I  L+
Sbjct: 69  KALQKLIKMNFLSKKRSEIDERTVVVYVNEQQRNSINEMIEALQ 112
```

tr Q4L437 Q4L437_STAHJ **Staphylococcal accessory regulator A [sarA] [Staphylococcus haemolyticus (strain JCSC1435)]** 124 AA align

```
 Score = 52.4 bits (124), Expect = 2e-06
 Identities = 31/114 (27%), Positives = 55/114 (48%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           +SKIND  +L+       K     KK+F++++EE  +L +I  +S+ +E   K+I     +
Sbjct: 3   ISKINDCFELLAMITYADKLKNIIKKEFSISFEEFAVLTYISQSKEDEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           K                     R+  DERTV++ V  TQ+  I  L++++   I+
Sbjct: 63  KQPQVVKAVKNLSQEDYFDKKRNEHDERTVLILVNSTQRKKIDSLLNKVNTRIE 116
```

tr Q49VG2 Q49VG2_STASA **Staphylococcal accessory regulator A [SSP2103] [Staphylococcus saprophyticus subsp. saprophyticus ATCC 15305]** 124 AA align

```
 Score = 52.4 bits (124), Expect = 2e-06
 Identities = 32/114 (28%), Positives = 53/114 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L+       K     KK+F++++EE  +L +I   ES E   K+I     +
Sbjct: 3   ITKINDCFELLAMVTYADKLKGIIKKEFSISFEEFAVLTYISEHESEEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           K                     R+  DERTV++ V   Q+  I +L+S + + IK
Sbjct: 63  KQPQVVKAVKNLSQEDYFDKKRNEHDERTVLILVNTKQRKKINELLSRVNDRIK 116
```

```
tr  Q6GJ52        Staphylococcal accessory regulator A [sarA]              124
    Q6GJ52_STAAR  [Staphylococcus aureus                                   AA
                  (strain MRSA252)]                                        align

 Score = 48.9 bits (115), Expect = 3e-05
 Identities = 29/113 (25%), Positives = 53/113 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L++       K     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ITKINDCFELLSMVTYADKLKSLIKKEFSISFEEFAVLTYISENKEKEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I+ L+S + + I
Sbjct: 63  KQPQVVKAVKILSQEDYFDKKRNEHDERTVLILVNAQQRKKIESLLSRVNKRI 115
```

```
tr  Q6GBL2        Staphylococcal accessory regulator A [SAS0584]           124
    Q6GBL2_STAAS  [Staphylococcus                                          AA
                  aureus (strain MSSA476)]                                 align

 Score = 48.9 bits (115), Expect = 3e-05
 Identities = 29/113 (25%), Positives = 53/113 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L++       K     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ITKINDCFELLSMVTYADKLKSLIKKEFSISFEEFAVLTYISENKEKEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I+ L+S + + I
Sbjct: 63  KQPQVVKAVKILSQEDYFDKKRNEHDERTVLILVNAQQRKKIESLLSRVNKRI 115
```

```
tr  Q7A732        Staphylococcal accessory regulator A [sarA]              124
    Q7A732_STAAN  [Staphylococcus aureus                                   AA
                  (strain N315)]                                           align

 Score = 48.9 bits (115), Expect = 3e-05
 Identities = 29/113 (25%), Positives = 53/113 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L++       K     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ITKINDCFELLSMVTYADKLKSLIKKEFSISFEEFAVLTYISENKEKEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I+ L+S + + I
Sbjct: 63  KQPQVVKAVKILSQEDYFDKKRNEHDERTVLILVNAQQRKKIESLLSRVNKRI 115
```

```
tr  Q7A2W5        Staphylococcal accessory regulator A [sarA]              124
    Q7A2W5_STAAM  [Staphylococcus aureus                                   AA
                  (strain Mu50 / ATCC 700699)]                             align

 Score = 48.9 bits (115), Expect = 3e-05
```

```
 Identities = 29/113 (25%), Positives = 53/113 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L++      K     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ITKINDCFELLSMVTYADKLKSLIKKEFSISFEEFAVLTYISENKEKEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I+ L+S + + I
Sbjct: 63  KQPQVVKAVKILSQEDYFDKKRNEHDERTVLILVNAQQRKKIESLLSRVNKRI 115
```

tr Q7A1N5 **Staphylococcal accessory regulator A [sarA]** 124
Q7A1N5_STAAW **[Staphylococcus aureus** AA
(strain MW2)] align

```
 Score = 48.9 bits (115), Expect = 3e-05
 Identities = 29/113 (25%), Positives = 53/113 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L++      K     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ITKINDCFELLSMVTYADKLKSLIKKEFSISFEEFAVLTYISENKEKEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I+ L+S + + I
Sbjct: 63  KQPQVVKAVKILSQEDYFDKKRNEHDERTVLILVNAQQRKKIESLLSRVNKRI 115
```

tr Q5HI51 **Staphylococcal accessory regulator A [sarA]** 124
Q5HI51_STAAC **[Staphylococcus aureus** AA
(strain COL)] align

```
 Score = 48.9 bits (115), Expect = 3e-05
 Identities = 29/113 (25%), Positives = 53/113 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L++      K     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ITKINDCFELLSMVTYADKLKSLIKKEFSISFEEFAVLTYISENKEKEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I+ L+S + + I
Sbjct: 63  KQPQVVKAVKILSQEDYFDKKRNEHDERTVLILVNAQQRKKIESLLSRVNKRI 115
```

tr Q53600 **Staphylococcal accessory regulator variant** 124
Q53600_STAAU **(Staphylococcal** AA
accessory regulator A) [sarA] [Staphylococcus aureus] align

```
 Score = 48.9 bits (115), Expect = 3e-05
 Identities = 29/113 (25%), Positives = 53/113 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L++      K     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ITKINDCFELLSMVTYADKLKSLIKKEFSISFEEFAVLTYISENKEKEYYLKDIINHLNY 62
```

```
Query: 61  KPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I+ L+S + + I
Sbjct: 63  KQPQVVKAVKILSQEDYFDKKRNEHDERTVLILVNAQQRKKIESLLSRVNKRI 115
```

tr Q53777 **SarA [sarA] [Staphylococcus aureus]** 113 AA
Q53777_STAAU align

```
 Score = 48.1 bits (113), Expect = 4e-05
 Identities = 28/111 (25%), Positives = 52/111 (46%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           ++KIND  +L++      K     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ITKINDCFELLSMVTYADKLKSLIKKEFSISFEEFAVLTYISENKEKEYYFKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEE 111
           K                     R+  DERTV++ V   Q+  I+ L+S + +
Sbjct: 63  KQPQVVKAVKILSQEDYFDKKRNEHDERTVLILVNAQQRKKIESLLSRVNK 113
```

tr Q5HRB9 **Staphylococcal accessory regulator A [sarA]** 124
Q5HRB9_STAEQ **[Staphylococcus** AA
epidermidis (strain ATCC 35984 / RP62A)] align

```
 Score = 45.1 bits (105), Expect = 4e-04
 Identities = 28/113 (24%), Positives = 49/113 (42%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           +SKIND  +L+       +     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ISKINDCFELLAMVTYADRLKGIIKKEFSISFEEFAVLTYISENKEEEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I  L+  +  I
Sbjct: 63  KQPQVVKAVKNLSQENYFNKKRNEHDERTVLILVDSKQRKKIDDLLKRVNNRI 115
```

tr O85233 **Staphylococcal accessory regulator A [sarA]** 124
O85233_STAEP **[Staphylococcus** AA
epidermidis] align

```
 Score = 45.1 bits (105), Expect = 4e-04
 Identities = 28/113 (24%), Positives = 49/113 (42%)

Query: 1   MSKINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           +SKIND  +L+       +     KK+F++++EE  +L +I  ++  E   K+I     +
Sbjct: 3   ISKINDCFELLAMVTYADRLKGIIKKEFSISFEEFAVLTYISENKEEEYYLKDIINHLNY 62

Query: 61  KPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           K                     R+  DERTV++ V   Q+  I  L+  +  I
Sbjct: 63  KQPQVVKAVKNLSQENYFNKKRNEHDERTVLILVDSKQRKKIDDLLKRVNNRI 115
```

tr Q5HNE7 Repressor of toxins [rot] [Staphylococcus epidermidis 136
Q5HNE7_STAEQ (strain ATCC AA
35984 / RP62A)] align

Score = 44.7 bits (104), Expect = 5e-04
Identities = 27/99 (27%), Positives = 46/99 (46%), Gaps = 2/99 (2%)

```
Query: 16  QVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXXXXXXXXXXXX 75
           ++   F   K+++ ++ EEI IL  +L  E   ++ KE+ K    KPY
Sbjct: 24  EIDSIFNTIKEEYGMSKEEILILLTLL--EKGSMTLKEMDKYVHIKPYKRTRTYNNLVNL 81

Query: 76  XXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
                R   DERTVI++  D Q +  + L+  +++ IK
Sbjct: 82  EWIYKERPQDDERTVIIHFNDKQNSKKEDLLKFIDDSIK 120
```

tr Q6G6H6 Putative staphylococcal accessory regulator [SAS2385] 247 AA
Q6G6H6_STAAS [Staphylococcus aureus (strain MSSA476)]
align

Score = 41.2 bits (95), Expect = 0.005
Identities = 27/108 (25%), Positives = 48/108 (44%)

```
Query: 7   INDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXX 66
           +N  +N    +    ++ K+++ L+ +E+ IL +      + IS KEI     +K
Sbjct: 9   VNKFINVEAYIFFLTQELKQQYKLSLKELLILAYFYYKNEHSISLKEIIGDILYKQSDVV 68

Query: 67  XXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
                        R+  DER + V VT  Q+  I  +I+EL++ IK
Sbjct: 69  KNIKSLSKKGFINKSRNEADERRIFVSVTPIQRKKIACVINELDKIIK 116
```

Score = 31.6 bits (70), Expect = 4.3
Identities = 22/91 (24%), Positives = 42/91 (45%), Gaps = 2/91 (2%)

```
Query: 25  KKKFNLNYEEIYILNHILRSESNEISS-KEIAKCSEFKPYYXXXXXXXXXXXXXXXXXRS 83
           K +FNL + ++ IL +++ S  NEI + K++ +   F                    RS
Sbjct: 152 KYRFNLTFLDLSIL-YVISSRKNEILNLKDLFESIRFMYPQIVRSVNRLNNKGMLIKERS 210

Query: 84  LQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           L DER V++ +   Q   I+ + + ++  + +K
Sbjct: 211 LADERIVLIKINKIQYNTIKSIFTDTSKILK 241
```

tr Q99RD5 Staphylococcal accessory regulator A homolog [sarH2] 247 AA
Q99RD5_STAAM [Staphylococcus aureus (strain Mu50 / ATCC 700699)]
align

Score = 41.2 bits (95), Expect = 0.005
Identities = 27/108 (25%), Positives = 48/108 (44%)

```
Query: 7   INDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXX 66
           +N  +N    +    ++ K+++ L+ +E+ IL +      + IS KEI     +K
Sbjct: 9   VNKFINVEAYIFFLTQELKQQYKLSLKELLILAYFYYKNEHSISLKEIIGDILYKQSDVV 68
```

```
Query: 67  XXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
                        R+  DER + V VT  Q+  I  +I+EL++ IK
Sbjct: 69  KNIKSLSKKGFINKSRNEADERRIFVSVTPIQRKKIACVINELDKIIK 116


 Score = 31.6 bits (70), Expect = 4.3
 Identities = 22/91 (24%), Positives = 42/91 (45%), Gaps = 2/91 (2%)

Query: 25  KKKFNLNYEEIYILNHILRSESNEISS-KEIAKCSEFKPYYXXXXXXXXXXXXXXXXXRS 83
           K +FNL + ++ IL +++ S  NEI + K++ +   F                      RS
Sbjct: 152 KYRFNLTFLDLSIL-YVISSRKNEILNLKDLFESIRFMYPQIVRSVNRLNNKGMLIKERS 210

Query: 84  LQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           L DER V++ +   Q   I+ + ++  + +K
Sbjct: 211 LADERIVLIKINKIQYNTIKSIFTDTSKILK 241
```

tr Q7A3K0 Q7A3K0_STAAN **SarH2 protein [sarH2] [Staphylococcus aureus (strain N315)]** 247 AA align

```
 Score = 41.2 bits (95), Expect = 0.005
 Identities = 27/108 (25%), Positives = 48/108 (44%)

Query: 7   INDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXX 66
           +N  +N    +    ++ K+++ L+ +E+ IL +      + IS KEI     +K
Sbjct: 9   VNKFINVEAYIFFLTQELKQQYKLSLKELLILAYFYYKNEHSISLKEIIGDILYKQSDVV 68

Query: 67  XXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
                        R+  DER + V VT  Q+  I  +I+EL++ IK
Sbjct: 69  KNIKSLSKKGFINKSRNEADERRIFVSVTPIQRKKIACVINELDKIIK 116


 Score = 31.6 bits (70), Expect = 4.3
 Identities = 22/91 (24%), Positives = 42/91 (45%), Gaps = 2/91 (2%)

Query: 25  KKKFNLNYEEIYILNHILRSESNEISS-KEIAKCSEFKPYYXXXXXXXXXXXXXXXXXRS 83
           K +FNL + ++ IL +++ S  NEI + K++ +   F                      RS
Sbjct: 152 KYRFNLTFLDLSIL-YVISSRKNEILNLKDLFESIRFMYPQIVRSVNRLNNKGMLIKERS 210

Query: 84  LQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           L DER V++ +   Q   I+ + ++  + +K
Sbjct: 211 LADERIVLIKINKIQYNTIKSIFTDTSKILK 241
```

tr Q7A004 Q7A004_STAAW **SarH2 protein [sarH2] [Staphylococcus aureus (strain MW2)]** 247 AA align

```
 Score = 41.2 bits (95), Expect = 0.005
 Identities = 27/108 (25%), Positives = 48/108 (44%)

Query: 7   INDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXX 66
           +N  +N    +    ++ K+++ L+ +E+ IL +      + IS KEI     +K
Sbjct: 9   VNKFINVEAYIFFLTQELKQQYKLSLKELLILAYFYYKNEHSISLKEIIGDILYKQSDVV 68
```

```
Query: 67  XXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
                         R+  DER + V VT  Q+  I  +I+EL++ IK
Sbjct: 69  KNIKSLSKKGFINKSRNEADERRIFVSVTPIQRKKIACVINELDKIIK 116
```

Score = 31.6 bits (70), Expect = 4.3
Identities = 22/91 (24%), Positives = 42/91 (45%), Gaps = 2/91 (2%)

```
Query: 25  KKKFNLNYEEIYILNHILRSESNEISS-KEIAKCSEFKPYYXXXXXXXXXXXXXXXXXRS 83
           K +FNL + ++ IL +++ S  NEI + K++ +   F                     RS
Sbjct: 152 KYRFNLTFLDLSIL-YVISSRKNEILNLKDLFESIRFMYPQIVRSVNRLNNKGMLIKERS 210

Query: 84  LQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           L DER V++ +   Q   I+ + ++  + +K
Sbjct: 211 LADERIVLIKINKIQYNTIKSIFTDTSKILK 241
```

tr Q5HD55 Q5HD55_STAAC **Staphylococcal accessory regulator U [sarU] [Staphylococcus aureus (strain COL)]** 247 AA align

Score = 41.2 bits (95), Expect = 0.005
Identities = 27/108 (25%), Positives = 48/108 (44%)

```
Query: 7   INDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXX 66
           +N  +N    +    ++ K+++ L+ +E+ IL +      + IS KEI     +K
Sbjct: 9   VNKFINVKAYIFFLTQELKQQYKLSLKELLILAYFYYKNEHSISLKEIIGDILYKQSDVV 68

Query: 67  XXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
                         R+  DER + V VT  Q+  I  +I+EL++ IK
Sbjct: 69  KNIKSLSKKGFINKSRNEADERRIFVSVTPIQRKKIACVINELDKIIK 116
```

Score = 31.6 bits (70), Expect = 4.3
Identities = 22/91 (24%), Positives = 42/91 (45%), Gaps = 2/91 (2%)

```
Query: 25  KKKFNLNYEEIYILNHILRSESNEISS-KEIAKCSEFKPYYXXXXXXXXXXXXXXXXXRS 83
           K +FNL + ++ IL +++ S  NEI + K++ +   F                     RS
Sbjct: 152 KYRFNLTFLDLSIL-YVISSRKNEILNLKDLFESIRFMYPQIVRSVNRLNNKGMLIKERS 210

Query: 84  LQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           L DER V++ +   Q   I+ + ++  + +K
Sbjct: 211 LADERIVLIKINKIQYNTIKSIFTDTSKILK 241
```

tr Q9EZK4 Q9EZK4_STAAU **Rot-like protein Rlp [rlp] [Staphylococcus aureus]** 247 AA align

Score = 41.2 bits (95), Expect = 0.005
Identities = 27/108 (25%), Positives = 48/108 (44%)

```
Query: 7   INDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXX 66
           +N  +N    +    ++ K+++ L+ +E+ IL +      + IS KEI     +K
Sbjct: 9   VNKFINVKAYIFFLTQELKQQYKLSLKELLILAYFYYKNEHSISLKEIIGDILYKQSDVV 68
```

```
Query: 67  XXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
                         R+  DER + V VT  Q+  I  +I+EL++ IK
Sbjct: 69  KNIKSLSKKGFINKSRNEADERRIFVSVTPIQRKKIACVINELDKIIK 116
```

Score = 31.6 bits (70), Expect = 4.3
Identities = 22/91 (24%), Positives = 42/91 (45%), Gaps = 2/91 (2%)

```
Query: 25  KKKFNLNYEEIYILNHILRSESNEISS-KEIAKCSEFKPYYXXXXXXXXXXXXXXXXXRS 83
           K +FNL + ++ IL +++ S  NEI + K++ +   F                     RS
Sbjct: 152 KYRFNLTFLDLSIL-YVISSRKNEILNLKDLFESIRFMYPQIVRSVNRLNNKGMLIKERS 210

Query: 84  LQDERTVIVYVTDTQKANIQKLISELEEYIK 114
           L DER V++ +   Q   I+ + ++  + +K
Sbjct: 211 LADERIVLIKINKIQYNTIKSIFTDTSKILK 241
```

tr Q8CNU6 Q8CNU6_STAEP **Repressor of toxins Rot [SE1435] [Staphylococcus epidermidis]** 99 AA align

Score = 37.7 bits (86), Expect = 0.060
Identities = 25/85 (29%), Positives = 39/85 (45%), Gaps = 2/85 (2%)

```
Query: 30  LNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXXXXXXXXXXXXXXXXXXRSLQDERT 89
           ++ EEI IL  +L  E   ++ KE+ K    KPY                  R   DERT
Sbjct: 1   MSKEEILILLTLL--EKGSMTLKEMDKYVHIKPYKRTRTYNNLVNLEWIYKERPQDDERT 58

Query: 90  VIVYVTDTQKANIQKLISELEEYIK 114
           VI++  D Q +  + L+  +++ IK
Sbjct: 59  VIIHFNDKQNSKKEDLLKFIDDSIK 83
```

tr Q6GKJ3 Q6GKJ3_STAAR **Putative regulatory protein [SAR0115] [Staphylococcus aureus (strain MRSA252)]** 250 AA align

Score = 35.8 bits (81), Expect = 0.23
Identities = 21/108 (19%), Positives = 45/108 (41%)

```
Query: 6   DINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYX 65
           D  + +N       F    KK   L++ E  IL  I     N +  K++ +    K
Sbjct: 132 DSKEFLNLMMYTMYFKNIIKKHLTLSFVEFTILAIITSQNKNIVLLKDLIETIHHKYPQT 191

Query: 66  XXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
                          RS +DER +++++ D Q+ + ++L++++ + +
Sbjct: 192 VRALNNLKKQGYLIKERSTEDERKILIHMNDAQQDHAEQLLAQVNQLL 239
```

sp Q7A0L8 ROT_STAAW **HTH-type transcriptional regulator rot (Repressor of toxins) [rot] [Staphylococcus aureus (strain MW2)]** 166 AA align

```
 Score = 35.4 bits (80), Expect = 0.30
 Identities = 29/120 (24%), Positives = 53/120 (44%), Gaps = 11/120 (9%)

Query: 1   MSKINDINDLVNATFQ-------VKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKE 53
           M K+N  ND V    Q       +   F + + ++ ++ EEI IL  + +  S  ++ KE
Sbjct: 34  MKKVN--NDTVFGILQLETLLGDINSIFSEIESEYKMSREEILILLTLWQKGS--MTLKE 89

Query: 54  IAKCSEFKPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           + +  E KPY                  R + DERTVI++  +  +    +L++ + + I
Sbjct: 90  MDRFVEVKPYKRTRTYNNLVELEWIYKERPVDDERTVIIHFNEKLQQEKVELLNFISDAI 149
```

sp Q6G8G5 ROT_STAAS **HTH-type transcriptional regulator rot (Repressor of toxins) [rot] [Staphylococcus aureus (strain MSSA476)]** 166 AA align

```
 Score = 35.4 bits (80), Expect = 0.30
 Identities = 29/120 (24%), Positives = 53/120 (44%), Gaps = 11/120 (9%)

Query: 1   MSKINDINDLVNATFQ-------VKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKE 53
           M K+N  ND V    Q       +   F + + ++ ++ EEI IL  + +  S  ++ KE
Sbjct: 34  MKKVN--NDTVFGILQLETLLGDINSIFSEIESEYKMSREEILILLTLWQKGS--MTLKE 89

Query: 54  IAKCSEFKPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           + +  E KPY                  R + DERTVI++  +  +    +L++ + + I
Sbjct: 90  MDRFVEVKPYKRTRTYNNLVELEWIYKERPVDDERTVIIHFNEKLQQEKVELLNFISDAI 149
```

sp Q6GFT9 ROT_STAAR **HTH-type transcriptional regulator rot (Repressor of toxins) [rot] [Staphylococcus aureus (strain MRSA252)]** 166 AA align

```
 Score = 35.4 bits (80), Expect = 0.30
 Identities = 29/120 (24%), Positives = 53/120 (44%), Gaps = 11/120 (9%)

Query: 1   MSKINDINDLVNATFQ-------VKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKE 53
           M K+N  ND V    Q       +   F + + ++ ++ EEI IL  + +  S  ++ KE
Sbjct: 34  MKKVN--NDTVFGILQLETLLGDINSIFSEIESEYKMSREEILILLTLWQKGS--MTLKE 89

Query: 54  IAKCSEFKPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           + +  E KPY                  R + DERTVI++  +  +    +L++ + + I
Sbjct: 90  MDRFVEVKPYKRTRTYNNLVELEWIYKERPVDDERTVIIHFNEKLQQEKVELLNFISDAI 149
```

sp Q7A514 ROT_STAAN **HTH-type transcriptional regulator rot (Repressor of toxins) [rot] [Staphylococcus aureus (strain N315)]** 166 AA align

```
 Score = 35.4 bits (80), Expect = 0.30
 Identities = 29/120 (24%), Positives = 53/120 (44%), Gaps = 11/120 (9%)

Query: 1   MSKINDINDLVNATFQ-------VKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKE 53
           M K+N  ND V    Q       +   F + + ++ ++ EEI IL  + +  S  ++ KE
```

```
Sbjct: 34  MKKVN--NDTVFGILQLETLLGDINSIFSEIESEYKMSREEILILLTLWQKGS--MTLKE 89

Query: 54  IAKCSEFKPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           + +  E KPY                   R + DERTVI++  +  +    +L++ + + I
Sbjct: 90  MDRFVEVKPYKRTRTYNNLVELEWIYKERPVDDERTVIIHFNEKLQQEKVELLNFISDAI 149
```

sp Q99TA4 ROT_STAAM **HTH-type transcriptional regulator rot (Repressor of toxins) [rot] [Staphylococcus aureus (strain Mu50 / ATCC 700699)]** 166 AA align

```
 Score = 35.4 bits (80), Expect = 0.30
 Identities = 29/120 (24%), Positives = 53/120 (44%), Gaps = 11/120 (9%)

Query: 1   MSKINDINDLVNATFQ-------VKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKE 53
           M K+N  ND V    Q       +   F + + ++ ++ EEI IL  + +  S  ++ KE
Sbjct: 34  MKKVN--NDTVFGILQLETLLGDINSIFSEIESEYKMSREEILILLTLWQKGS--MTLKE 89

Query: 54  IAKCSEFKPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           + +  E KPY                   R + DERTVI++  +  +    +L++ + + I
Sbjct: 90  MDRFVEVKPYKRTRTYNNLVELEWIYKERPVDDERTVIIHFNEKLQQEKVELLNFISDAI 149
```

tr Q6GD13 Q6GD13_STAAS **Putative regulatory protein [SAS0086] [Staphylococcus aureus (strain MSSA476)]** 250 AA align

```
 Score = 35.4 bits (80), Expect = 0.30
 Identities = 21/108 (19%), Positives = 45/108 (41%)

Query: 6   DINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYX 65
           D  + +N       F    KK   L++ E  IL  I     N +  K++ +    K
Sbjct: 132 DSKEFLNLMMYTMYFKNIIKKHLTLSFVEFTILAIITSQNKNIVLLKDLIETIHHKYPQT 191

Query: 66  XXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
                        RS +DER +++++ D Q+ + ++L+++ + +
Sbjct: 192 VRALNNLKKQGYLIKERSTEDERKILIHMDDAQQDHAEQLLAQVNQLL 239
```

tr Q7A872 Q7A872_STAAN **SarH1 protein [sarH1] [Staphylococcus aureus (strain N315)]** 250 AA align

```
 Score = 35.4 bits (80), Expect = 0.30
 Identities = 21/108 (19%), Positives = 45/108 (41%)

Query: 6   DINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYX 65
           D  + +N       F    KK   L++ E  IL  I     N +  K++ +    K
Sbjct: 132 DSKEFLNLMMYTMYFKNIIKKHLTLSFVEFTILAIITSQNKNIVLLKDLIETIHHKYPQT 191

Query: 66  XXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
                        RS +DER +++++ D Q+ + ++L+++ + +
Sbjct: 192 VRALNNLKKQGYLIKERSTEDERKILIHMDDAQQDHAEQLLAQVNQLL 239
```

tr Q7A2Y8 Staphylococcal accessory regulator A homologue [sarH1] 250 AA
Q7A2Y8_STAAM [Staphylococcus aureus (strain Mu50 / ATCC 700699)]
align

Score = 35.4 bits (80), Expect = 0.30
Identities = 21/108 (19%), Positives = 45/108 (41%)

```
Query: 6   DINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYX 65
           D  + +N        F    KK   L++ E  IL  I     N +  K++ +    K
Sbjct: 132 DSKEFLNLMMYTMYFKNIIKKHLTLSFVEFTILAIITSQNKNIVLLKDLIETIHHKYPQT 191

Query: 66  XXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
                           RS +DER +++++ D Q+ + ++L++++ + +
Sbjct: 192 VRALNNLKKQGYLIKERSTEDERKILIHMDDAQQDHAEQLLAQVNQLL 239
```

tr Q7A1Z8 SarH1 protein [sarH1] [Staphylococcus aureus (strain MW2)] 250 AA
Q7A1Z8_STAAW
align

Score = 35.4 bits (80), Expect = 0.30
Identities = 21/108 (19%), Positives = 45/108 (41%)

```
Query: 6   DINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYX 65
           D  + +N        F    KK   L++ E  IL  I     N +  K++ +    K
Sbjct: 132 DSKEFLNLMMYTMYFKNIIKKHLTLSFVEFTILAIITSQNKNIVLLKDLIETIHHKYPQT 191

Query: 66  XXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
                           RS +DER +++++ D Q+ + ++L++++ + +
Sbjct: 192 VRALNNLKKQGYLIKERSTEDERKILIHMDDAQQDHAEQLLAQVNQLL 239
```

tr Q5HJQ7 Staphylococcal accessory regulator S [sarS] 250 AA
Q5HJQ7_STAAC [Staphylococcus aureus (strain COL)]
align

Score = 35.4 bits (80), Expect = 0.30
Identities = 21/108 (19%), Positives = 45/108 (41%)

```
Query: 6   DINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYX 65
           D  + +N        F    KK   L++ E  IL  I     N +  K++ +    K
Sbjct: 132 DSKEFLNLMMYTMYFKNIIKKHLTLSFVEFTILAIITSQNKNIVLLKDLIETIHHKYPQT 191

Query: 66  XXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
                           RS +DER +++++ D Q+ + ++L++++ + +
Sbjct: 192 VRALNNLKKQGYLIKERSTEDERKILIHMDDAQQDHAEQLLAQVNQLL 239
```

tr Q9KWJ2 Hyothetical protein (SarH1) [orfX] [Staphylococcus 250

Q9KWJ2_STAAU aureus] AA align

Score = 35.4 bits (80), Expect = 0.30
Identities = 21/108 (19%), Positives = 45/108 (41%)

```
Query: 6   DINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYX 65
           D  + +N       F    KK   L++ E  IL  I     N +  K++ +    K
Sbjct: 132 DSKEFLNLMMYTMYFKNIIKKHLTLSFVEFTILAIITSQNKNIVLLKDLIETIHHKYPQT 191

Query: 66  XXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
                          RS +DER +++++ D Q+ + ++L++++ + +
Sbjct: 192 VRALNNLKKQGYLIKERSTEDERKILIHMDDAQQDHAEQLLAQVNQLL 239
```

tr Q891H0 Q891H0_CLOTE **Hypothetical protein CTC02406 [CTC02406] [Clostridium tetani]** 149 AA align

Score = 34.7 bits (78), Expect = 0.51
Identities = 23/97 (23%), Positives = 42/97 (42%), Gaps = 1/97 (1%)

```
Query: 18  KKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKPYYXXXXXXXXXXXXXX 77
           KK+   +   FNL   EI IL+ ++ +   +I++ EI+ C
Sbjct: 22  KKYMEKSLSGFNLIPAEIDILSFLVNNIEKDITASEISMCRGISKGLVSRAVHSLKTKNV 81

Query: 78  XXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYIK 114
                + QD R+V + + D + + I+K + E+ E  K
Sbjct: 82  IELKENPQDGRSVYIKIVDEEDSLIKK-VKEINEKFK 117
```

sp Q9RFJ6 ROT_STAAU **HTH-type transcriptional regulator rot (Repressor of toxins) [rot] [Staphylococcus aureus]** 166 AA align

Score = 34.3 bits (77), Expect = 0.67
Identities = 28/120 (23%), Positives = 52/120 (43%), Gaps = 11/120 (9%)

```
Query: 1   MSKINDINDLVNATFQ-------VKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKE 53
           M K+N  ND V    Q       +   F + + ++ ++ EEI IL  +   +   ++ KE
Sbjct: 34  MKKVN--NDTVFGILQLETLLGDINSIFSEIESEYKMSREEILIL--LTLWQKGFMTLKE 89

Query: 54  IAKCSEFKPYYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           + +  E KPY                  R + DERTVI++  +  +    +L++ + + I
Sbjct: 90  MDRFVEVKPYKRTRTYNNLVELEWIYKERPVDDERTVIIHFNEKLQQEKVELLNFISDAI 149
```

sp Q5HF12 ROT_STAAC **HTH-type transcriptional regulator rot (Repressor of toxins) [rot] [Staphylococcus aureus (strain COL)]** 166 AA align

Score = 34.3 bits (77), Expect = 0.67
Identities = 28/120 (23%), Positives = 52/120 (43%), Gaps = 11/120 (9%)

```
Query: 1   MSKINDINDLVNATFQ-------VKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKE 53
           M K+N  ND V    Q       +   F + + ++ ++ EEI IL  +   +   ++ KE
Sbjct: 34  MKKVN--NDTVFGILQLETLLGDINSIFSEIESEYKMSREEILIL--LTLWQKGFMTLKE 89

Query: 54  IAKCSEFKPYYXXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           + +  E KPY                   R + DERTVI++  +  +    +L++ + + I
Sbjct: 90  MDRFVEVKPYKRTRTYNNLVELEWIYKERPVDDERTVIIHFNEKLQQEKVELLNFISDAI 149
```

tr Q49YJ2 Q49YJ2_STASA **Repressor of toxins [SSP1002] [Staphylococcus saprophyticus subsp. saprophyticus ATCC 15305]** 133 AA align

```
 Score = 34.3 bits (77), Expect = 0.67
 Identities = 25/111 (22%), Positives = 48/111 (42%), Gaps = 2/111 (1%)

Query: 3   KINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEFKP 62
           K++ +  L +    ++  F   +K++ ++ EEI IL  + +  S  ++ KE+       K
Sbjct: 8   KVDGVLQLESVLKDIEDIFDKVQKQYKMSKEEILILLTLWKEGS--MTLKEMDDFVHIKS 65

Query: 63  YYXXXXXXXXXXXXXXXXRSLQDERTVIVYVTDTQKANIQKLISELEEYI 113
           Y                   R   DERTVI++  +  K   + L++  +E I
Sbjct: 66  YKRTRTYNDLVEKAWIIKERPQNDERTVIIHFNEDLKDQRESLLNFFKEEI 116
```

tr Q7RHA3 Q7RHA3_PLAYO **Hypothetical protein (Fragment) [PY04086] [Plasmodium yoelii yoelii]** 630 AA align

```
 Score = 32.7 bits (73), Expect = 1.9
 Identities = 18/55 (32%), Positives = 28/55 (50%)

Query: 6   DINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKCSEF 60
           DI D     F     F  T   FNLNY    ++N++   +SN +  KE++K +E+
Sbjct: 367 DIYDFSYKIFDPICNFLHTYNCFNLNYISNKLINNLRAKKSNGVIKKELSKLTEY 421
```

tr Q8I3P4 Q8I3P4_PLAF7 **Hypothetical protein PFE1095w [PFE1095w] [Plasmodium falciparum (isolate 3D7)]** 1777 AA align

```
 Score = 32.3 bits (72), Expect = 2.5
 Identities = 17/56 (30%), Positives = 29/56 (51%), Gaps = 7/56 (12%)

Query: 5    NDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILN-------HILRSESNEISSKE 53
            NDIN+ V    Q ++ F + KK F     E+++L        +I++   NE++ KE
Sbjct: 1289 NDINEKVKLLKQREEIFYEEKKNFEKEKNELHLLKENVLNKMNIIKDRENELNKKE 1344
```

tr Q91G55 **043L [Chilo iridescent virus (CIV) (Insect iridescent** 116

Q91G55_IRV6 **virus type 6)]** AA align

Score = 30.8 bits (68), Expect = 7.4
Identities = 16/51 (31%), Positives = 30/51 (58%), Gaps = 8/51 (15%)

```
Query: 9  DLVN--ATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESNEISSKEIAKC 57
          DL+N     +++KF  D +KK+N+NY      N+++    N+ S++ + KC
Sbjct: 2  DLINNKLNIEIQKFCLDLEKKYNINY------NNLIDLWFNKESTERLIKC 46
```

tr Q4YX98 Q4YX98_PLABE **Hypothetical protein (Fragment) [PB000235.02.0] [Plasmodium berghei]** 436 AA align

Score = 30.8 bits (68), Expect = 7.4
Identities = 15/44 (34%), Positives = 22/44 (49%), Gaps = 1/44 (2%)

```
Query: 5   NDINDLVNATFQVKKFFRDTKKKFNLNYE-EIYILNHILRSESN 47
           NDIN  +N TF +K+ +   KK+F   Y   +Y   H    + N
Sbjct: 77  NDINKTINDTFFIKQVYESIKKRFKYIYNINLYEYMHSFNLKKN 120
```

tr Q513A0 Q513A0_ENTHI **Hypothetical protein [87.t00006] [Entamoeba histolytica HM-1:IMSS]** 725 AA align

Score = 30.4 bits (67), Expect = 9.6
Identities = 19/55 (34%), Positives = 29/55 (52%), Gaps = 1/55 (1%)

```
Query: 3   KINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILN-HILRSESNEISSKEIAK 56
           KI  +++L     +++K   +T+KKFN   EE+   N  I +    EI  KE AK
Sbjct: 177 KIEGVDELKCQVVKIQKTKEETEKKFNKEIEELKQTNREISKKLEAEIKKKEDAK 231
```

tr Q512X9 Q512X9_ENTHI **Hypothetical protein [87.t00028] [Entamoeba histolytica HM-1:IMSS]** 1011 AA align

Score = 30.4 bits (67), Expect = 9.6
Identities = 19/55 (34%), Positives = 29/55 (52%), Gaps = 1/55 (1%)

```
Query: 3   KINDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILN-HILRSESNEISSKEIAK 56
           KI  +++L     +++K   +T+KKFN   EE+   N  I +    EI  KE AK
Sbjct: 332 KIEGVDELKCQVVKIQKTKEETEKKFNKEIEELKQTNREISKKLEAEIKKKEDAK 386
```

tr Q4Z0B5 Q4Z0B5_PLABE **Hypothetical protein [PB103359.00.0] [Plasmodium berghei]** 1043 AA align

Score = 30.4 bits (67), Expect = 9.6

```
 Identities = 18/61 (29%), Positives = 37/61 (60%), Gaps = 5/61 (8%)

Query: 1    MSKINDINDLVNATFQ---VKKFFRDTKKKFNLNYEEIYILNHILRSESN--EISSKEIA 55
            ++K +DI+  +  TF+    +K+F +T KK ++ YEEI+ +N    +  N    ++K++A
Sbjct: 975  LNKKDDISCKIKKTFKKNKYEKYFEETFKKEHMLYEEIFEINERDNNMENGESCTNKQVA 1034

Query: 56   K 56
            +
Sbjct: 1035 Q 1035
```

tr Q4XQR2 Q4XQR2_PLACH **Hypothetical protein (Fragment) [PC000212.04.0] [Plasmodium chabaudi]** 503 AA align

```
 Score = 30.4 bits (67), Expect = 9.6
 Identities = 17/67 (25%), Positives = 31/67 (45%), Gaps = 13/67 (19%)

Query: 5   NDINDLVNATFQVKKFFRDTKKKFNLNYEEIYILNHILRSESN----------EISSKEI 54
           N+ N ++N  F+ +K    +     LNY+E+   N++  +E N           +S++ I
Sbjct: 427 NNTNSIINTFFKNEKTIEQSSSNIFLNYDEV---NNLKENEDNFKNGNGSKISSLSNENI 483

Query: 55  AKCSEFK 61
            KC   K
Sbjct: 484 GKCYNIK 490
```

```
Database: EXPASY/UniProtKB
    Posted date:  Oct 10, 2005 11:17 AM
  Number of letters in database: 793,847,190
  Number of sequences in database:  2,420,647

Lambda     K      H
   0.315    0.132    0.351

Gapped
Lambda     K      H
   0.267   0.0410    0.140


Matrix: BLOSUM62
Gap Penalties: Existence: 11, Extension: 1
length of query: 115
length of database: 793,847,190
effective HSP length: 91
effective length of query: 24
effective length of database: 573,568,313
effective search space: 13765639512
effective search space used: 13765639512
T: 11
A: 40
X1: 16 ( 7.3 bits)
X2: 38 (14.6 bits)
X3: 64 (24.7 bits)
S1: 41 (21.6 bits)
S2: 67 (30.4 bits)
```

Wallclock time: 2 seconds

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Search Results - Record(s) 1 through 4 of 4 returned.

L2: Entry 1 of 4 File: USPT Nov 2, 1999

US-PAT-NO: 5976792
DOCUMENT-IDENTIFIER: US 5976792 A

TITLE: Regulation of exoprotein in staphylococcus aureus

DATE-ISSUED: November 2, 1999

US-CL-CURRENT: 435/6; 435/320.1, 530/350, 530/387.1, 530/388.1, 530/388.4, 530/825, 536/23.7, 536/24.32

INT-CL: [06] C12 Q 1/68, C07 H 21/04, C12 N 15/74, C07 K 14/31, C07 K 16/12

L2: Entry 2 of 4 File: USPT Dec 24, 1996

US-PAT-NO: 5587288
DOCUMENT-IDENTIFIER: US 5587288 A
**** See image for Certificate of Correction ****

TITLE: Regulation of exoprotein in Staphylococcus aureus

DATE-ISSUED: December 24, 1996

US-CL-CURRENT: 435/6; 530/350, 536/23.1, 536/24.3

INT-CL: [06] C12 Q 1/68, C07 K 13/00, C07 H 21/02, C07 H 21/04

L2: Entry 3 of 4 File: DWPI Nov 2, 1999

DERWENT-ACC-NO: 2000-021938
ABSTRACTED-PUB-NO: US 5976792A

TITLE: New accessory regulatory protein, sar, from Staphylococcus aureus, used to design analogs potentially useful as antibacterial agents

INT-CL (IPC): C07 H 21/04, C07 K 14/31, C07 K 16/12, C12 N 15/74, C12 Q 1/68
Derwent-CL (DC): B04, D16
CPI Codes: B04-E02B; B04-E03B; B04-E05; B04-E08; B04-F0100E; B04-G07; B04-N03A; B04-N03A0E; B11-C08E3; B11-C08E5; B12-K04A4; B12-K04F; D05-H04; D05-H09; D05-H11; D05-H12A; D05-H12B2; D05-H12E; D05-H17A2; D05-H17B2;

L2: Entry 4 of 4 File: DWPI Dec 24, 1996

DERWENT-ACC-NO: 1997-064792
ABSTRACTED-PUB-NO: US 5587288A

TITLE: New isolated staphylococcal accessory regulatory protein and gene - used to

develop prods. for use as antimicrobial agents and for detection of pathogenic staphylococci.

INT-CL (IPC): C07 H 21/02, C07 H 21/04, C07 K 13/00, C12 Q 1/68
Derwent-CL (DC): B04, D16
CPI Codes: B04-E01; B04-F10B3; B04-N02; B12-K04A; D05-C12; D05-H04; D05-H12A; D05-H17A6;

Previous Page Next Page

```
SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1951-2005/Oct 11
         (c) format only 2005 Dialog
  File   5:Biosis Previews(R) 1969-2005/Oct W2
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         (c) 2005 The HW Wilson Co.
  File 135:NewsRx Weekly Reports 1995-2005/Oct W1
         (c) 2005 NewsRx
*File 135: New newsletters are now added. See Help News135 for the
complete list of newsletters.
  File 144:Pascal 1973-2005/Oct W1
         (c) 2005 INIST/CNRS
  File 149:TGG Health&Wellness DB(SM) 1976-2005/Oct W1
         (c) 2005 The Gale Group
  File 156:ToxFile 1965-2005/Oct W2
         (c) format only 2005 Dialog
  File 159:Cancerlit 1975-2002/Oct
         (c) format only 2002 Dialog
*File 159: Cancerlit is no longer updating.
Please see HELP NEWS159.
  File 162:Global Health 1983-2005/Sep
         (c) 2005 CAB International
  File 164:Allied & Complementary Medicine 1984-2005/Oct
         (c) 2005 BLHCIS
  File 172:EMBASE Alert 2005/Oct 12
         (c) 2005 Elsevier Science B.V.
  File 266:FEDRIP 2005/Jun
        Comp & dist by NTIS, Intl Copyright All Rights Res
  File 369:New Scientist 1994-2005/Jul W1
         (c) 2005 Reed Business Information Ltd.
  File 370:Science 1996-1999/Jul W3
         (c) 1999 AAAS
*File 370: This file is closed (no updates). Use File 47 for more current
information.
  File 399:CA SEARCH(R) 1967-2005/UD=14316
         (c) 2005 American Chemical Society
*File 399: Use is subject to the terms of your user/customer agreement.
Alert feature enhanced for multiple files, etc. See HELP ALERT.
  File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
         (c) 1998 Inst for Sci Info
  File 444:New England Journal of Med. 1985-2005/Sep W4
         (c) 2005 Mass. Med. Soc.
  File 467:ExtraMED(tm) 2000/Dec
```

DIALOG Expand

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ef    Items  Index-term
E1        1  SAR CHELATE
E2        8  SAR PROTEIN, LYCOPERSICON ESCULENTUM
E3      301 *SARA
E4        1  SARA B
E5       64  SARA PROTEIN, BACTERIAL
E6        1  SARA PROTEIN, FUNGAL
E7        2  SARA PROTEIN, MOUSE
E8        5  SARA PROTEIN, STAPHYLOCOCCUS AUREUS
E9        1  SARA VON WURZBURG
E10       1  SARABANDE
E11       1  SARABHAI
E12       5  SARABIA

          Enter P or PAGE for more
? e sarr

Ref    Items  Index-term
E1        1  SARQAQ
E2        1  SARQI
E3       12 *SARR
E4        1  SARR M
E5        2  SARRA
E6        1  SARRABUS
E7        8  SARRACENIA
E8        2  SARRACENIACEAE
E9        1  SARRACENIAPURPUREA
E10       1  SARRACENINE
E11       1  SARRACENIUS
E12       1  SARRACENO
```

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*File 467: F467 no longer updates; see Help News467. 7.

Set	Items	Description

Cost is in DialUnits
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Terminal set to DLINK
? t s7/9/all

Set	Items	Description
S1	4792	P2 (2N) PROMOTER?
S2	492264	STAPHY?
S3	116	S1 AND S2
S4	69	S3 AND (SAR OR SARR OR SARA?)
S5	19	S4/2001:2005
S6	50	S4 NOT S5
S7	12	RD (unique items)

? logoff hold

7/9/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13446285 PMID: 10411747
Characterization of the SarA virulence gene regulator of Staphylococcus aureus.
Rechtin T M; Gillaspy A F; Schumacher M A; Brennan R G; Smeltzer M S; Hurlburt B K
Department of Biochemistry and Molecular Biology, University of Arkansas for Medical Sciences, Little Rock 72205, USA.
Molecular microbiology (ENGLAND) Jul 1999, 33 (2) p307-16, ISSN 0950-382X Journal Code: 8712028
Contract/Grant No.: AI43356; AI; NIAID
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
Staphylococcus aureus is a potent human pathogen that expresses a large number of virulence factors in a temporally regulated fashion. Two pleiotropically acting regulatory loci were identified in previous mutational studies. The agr locus comprises two operons that express a quorum-sensing system from the **P2 promoter** and a regulatory RNA molecule from the P3 promoter. The **sar** locus encodes a DNA-binding protein that activates the expression of both agr operons. We have cloned the **sarA** gene, expressed **SarA** in Escherichia coli and purified the recombinant protein to apparent homogeneity. The purified protein was found to be dimeric in the presence and absence of DNA and to consist mostly of alpha-helices. DNase I footprinting of **SarA** on the putative regulatory region cis to the agr promoters revealed three high-affinity binding sites composed of two half-sites each. Quantitative electrophoretic mobility shift assays (EMSAs) were used to derive equilibrium binding constants (KD) for the interaction of **SarA** with these binding sites. An unusual ladder banding pattern was observed in EMSA with a large DNA fragment including all three binding sites. Our data indicate that **SarA** regulation of the agr operons involves binding to multiple half-sites and may involve other sites located downstream of the promoters.
Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't,

P.H.S.
Descriptors: *Bacterial Proteins--metabolism--ME; *DNA-Binding Proteins --metabolism--ME; *Gene Expression Regulation, Bacterial; * **Staphylococcus** aureus--genetics--GE; *Trans-Activators; *Transcription Factors --metabolism--ME; Bacterial Proteins--genetics--GE; Base Sequence; Binding Sites; DNA Mutational Analysis; DNA-Binding Proteins--genetics--GE; Molecular Sequence Data; Protein Binding; RNA--metabolism--ME; RNA, Antisense--metabolism--ME; RNA, Bacterial--metabolism--ME; Recombinant Fusion Proteins--metabolism--ME; **Staphylococcus** aureus--pathogenicity--PY ; Transcription, Genetic; Virulence--genetics--GE
CAS Registry No.: 0 (Agr protein, Staphylococcus aureus); 0 (Bacterial Proteins); 0 (DNA-Binding Proteins); 0 (RNA primers); 0 (RNA, Antisense); 0 (RNA, Bacterial); 0 (RNAIII); 0 (Recombinant Fusion Proteins); 0 (SarA protein, bacterial); 0 (Trans-Activators); 0 (Transcription Factors); 63231-63-0 (RNA)
Record Date Created: 19980223
Record Date Completed: 19980223

7/9/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12039726 PMID: 9335283
Alternative transcription factor sigmaSB of Staphylococcus **aureus: characterization and role in transcription of the global regulatory locus** sar .
Deora R; Tseng T; Misra T K
Department of Microbiology and Immunology, University of Illinois College of Medicine, Chicago 60612, USA.
Journal of bacteriology (UNITED STATES) Oct 1997, 179 (20) p6355-9, ISSN 0021-9193 Journal Code: 2985120R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
A homolog of the multiple-stress-responsive transcription factor sigmaB of Bacillus subtilis was predicted from the DNA sequence analysis of a region of the **Staphylococcus** aureus chromosome. A hybrid between the coding sequence of the first 11 amino acids of the gene 10 leader peptide of phage T7 (T7.Tag) and the putative sigB gene of S. aureus was constructed and cloned into Escherichia coli BL21(DE3)pLysS for overexpression from a T7 promoter. A homogeneous preparation of the overproduced protein was obtained by affinity chromatography with a T7.Tag monoclonal antibody coupled to agarose. The amino-terminal amino acid sequence of the first 22 residues of the purified protein matched that deduced from the nucleotide sequence. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis of the purified protein, designated sigmaSB, indicated that it migrated as an approximately 39-kDa polypeptide. Promoter-specific transcription from the B. subtilis sigmaB-dependent PB promoter of the sigB operon was stimulated by sigmaSB in a concentration-dependent fashion when reconstituted with the S. aureus core RNA polymerase (RNAP). Specific transcript from the predicted sigmaB-dependent PB promoter of the sigB operon of S. aureus was obtained by the reconstituted RNAP in a runoff transcription reaction. The **sar** operon of S. aureus contains three **promoter** elements (P1, **P2** , and P3) and is known to partly control the synthesis of a number of extracellular toxins and several cell wall proteins. Our in vitro studies revealed that transcription from the P1

P.H.S.
Descriptors: *Bacterial Proteins--genetics--GE; *Gene Expression Regulation, Bacterial; * **Staphylococcus** aureus--pathogenicity--PY; *Trans-Activators; Base Sequence; Circular Dichroism; DNA Footprinting; Dimerization; Molecular Sequence Data; Protein Conformation; **Staphylococcus** aureus--genetics--GE
CAS Registry No.: 0 (Bacterial Proteins); 0 (SarA protein, bacterial); 0 (Trans-Activators)
Record Date Created: 19990902
Record Date Completed: 19990902

7/9/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12515108 PMID: 9826382
Selective activation of sar promoters with the use of green fluorescent protein transcriptional fusions as the detection system in the rabbit endocarditis model.
Cheung A L; Nast C C; Bayer A S
Laboratory of Bacterial Pathogenesis and Immunology, The Rockefeller University, New York, New York 10021, Los Angeles, California 90024, USA. cheunga@Rockvax.rockefeller.edu
Infection and immunity (UNITED STATES) Dec 1998, 66 (12) p5988-93, ISSN 0019-9567 Journal Code: 0246127
Contract/Grant No.: AI30061; AI; NIAID; AI37142; AI; NIAID; AI39108; AI; NIAID
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
The global regulatory locus **sar** is composed of three overlapping transcripts initiated from a triple-promoter system (designated P1, P3, and P2). To explore if the individual **sar** promoters are differentially expressed in vitro and in vivo, we constructed a shuttle plasmid (pALC1434) containing a promoterless gfpUV gene (a gfp derivative [Clontech]) preceded by a polylinker region. Recombinant shuttle vectors containing individual **sar** promoters upstream of the gfpUV reporter gene were then introduced into **Staphylococcus** aureus RN6390. Northern and immunoblot analysis revealed that P1 is stronger than the **P2** and P3 **promoters** in vitro. Additionally, the levels of the gfpUV transcript driven by individual **sar** promoters also correlated with the growth cycle dependency of these promoters in liquid cultures, thus suggesting the utility of pALC1434 as a vehicle for reporter fusion. Using the rabbit endocarditis model, we examined the expression of these three GFPUV fusions in vivo by fluorescence microscopy of infected cardiac vegetations 24 h after initial intravenous challenge. Similar to the in vitro findings, P1 was activated both in the center and on the surface of the vegetations. In contrast, the P3 promoter was silent both in vivo and in vitro as determined by fluorescence microscopy. Remarkably, P2 was silent in vitro but became highly activated in vivo. In particular, the **sar P2 promoter** was activated on the surface of the vegetation but not in the center of the lesion. These data imply that in vivo promoter activation of **sar** differed from that observed in vitro. Moreover, the individual **sar** promoters may be differentially expressed in different areas within the same anatomic niche, presumably reflecting the microbial physiological response to distinct host microenvironments. As the **sar** locus controls the synthesis

Taken together, these results indicate that the **sarA** -encoded protein, possibly in conjunction with peptides encoded in the upstream region, regulates hemolysin production by controlling agr P2 and P3 transcription.

Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Bacterial Proteins--genetics--GE; *Genes, Bacterial; * **Staphylococcus** aureus--genetics--GE; *Trans-Activators; *Transcription Factors--genetics--GE; *Transcription, Genetic; Amino Acid Sequence; Chromosome Mapping; Molecular Sequence Data

CAS Registry No.: 0 (Agr protein, Staphylococcus aureus); 0 (Bacterial Proteins); 0 (SarA protein, bacterial); 0 (Trans-Activators); 0 (Transcription Factors)

Record Date Created: 19970703

Record Date Completed: 19970703

7/9/7 (Item 7 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

11587547 PMID: 8898391

Transcriptional control of the agr-dependent virulence gene regulator, RNAIII, in Staphylococcus **aureus.**

Morfeldt E; Tegmark K; Arvidson S

Microbiology and Tumorbiology Center, Karolinska Institute, Stockholm, Sweden. evamorfeldt@mtc.ki.se

Molecular microbiology (ENGLAND) Sep 1996, 21 (6) p1227-37, ISSN 0950-382X Journal Code: 8712028

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Many of the genes coding for extracellular toxins, enzymes and cell-surface proteins in **Staphylococcus** aureus are regulated by a 510 nt RNA molecule, RNAIII. Expression of the RNAIII gene is positively controlled by the closely linked agr operon, which encodes a multicomponent signal-transduction system, and by an unlinked operon called **sar** . We have analysed the 120 bp region that separates the RNAIII promoter, P3, from the divergent agr **promoter** , **P2** . By transcription analysis, it was shown that P3 can function in trans of the agr operon. A stretch of 53 bp upstream of P3, containing an interrupted repeat of 7 bp, was found to be required for the agr-dependent expression of RNAIII. A single cytoplasmic protein was shown to bind to at least two sites within this regulatory region. The protein, which was absent in extracts from a **sarA** mutant, was identified as the **sarA** product by N-terminal amino acid sequencing. A DNA fragment from the P2 region, encompassing an almost identical repeated DNA motif, competed for the same protein. No interaction between the regulatory DNA sequence and any agr-dependent products could be demonstrated. The results of this study suggest that P3 and P2 are controlled by a mechanism involving the binding of the **SarA** protein to multiple sites within the regulatory regions immediately upstream of each promoter, and the as yet unknown activity of AgrA.

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Gene Expression Regulation, Bacterial; *RNA, Antisense --genetics--GE; *RNA, Bacterial--genetics--GE; * **Staphylococcus** aureus --genetics--GE; *Transcription, Genetic; Base Sequence; Molecular Sequence Data; Sequence Alignment; **Staphylococcus** aureus--pathogenicity--PY; Virulence--genetics--GE

CAS Registry No.: 0 (RNA, Antisense); 0 (RNA, Bacterial); 0 (RNAIII)
Record Date Created: 19970203
Record Date Completed: 19970203

7/9/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11442078 PMID: 8755885
The molecular architecture of the sar **locus in** Staphylococcus **aureus.**
Bayer M G; Heinrichs J H; Cheung A L
Laboratory of Bacterial Pathogenesis and Immunology, The Rockefeller University, New York, 10021, USA.
Journal of bacteriology (UNITED STATES) Aug 1996, 178 (15) p4563-70, ISSN 0021-9193 Journal Code: 2985120R
Contract/Grant No.: AI30061; AI; NIAID; AI37142; AI; NIAID
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
The global regulator **sar** in **Staphylococcus** aureus controls the synthesis of a variety of cell wall and extracellular proteins, many of which are putative virulence factors. The **sar** locus in strain RN6390 contains a 339-bp open reading frame (**sarA**) and an 860-bp upstream region. Transcriptional analyses of this locus revealed three different transcripts of 0.58, 0.84, and 1.15 kb (designated **sarA** , sarC, and sarB, respectively). All three transcripts seemed to be under temporal, growth cycle-dependent regulation, with **sarA** and sarB being most abundant in early log phase and the sarC concentration being highest toward the late stationary phase. Mapping of the 5' ends of the **sar** transcripts by primer extension and modified S1 nuclease protection assays demonstrated that transcription is initiated from three separate, widely spaced promoters. The 3' ends of all three **sar** transcripts are identical, and transcriptional termination occurs upstream of a typical prokaryotic poly(T) termination signal. Northern (RNA) analysis of **sar** mutant clones containing plasmids that comprised various promoters and the termination signal revealed that individual transcripts can be generated from each of the three promoters, thus suggesting possible activation as independent promoters. The multipromoter system, from which transcription is initiated, bears conserved features for recognition by homologous sigma 70 transcription factors and also by those expressed in the general stress response. Downstream of the two distal **promoters** (P3 and **P2**) are two regions potentially encoding short peptides. It is conceivable that posttranslational cooperation between these short peptides and the **sarA** gene product occurs to modulate **sar** -related functions. Complementation studies of a **sar** mutant with a clone expressing all three **sar** transcripts showed that this clone was able to restore the **sar** wild-type phenotype to the **sar** mutant.
Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Descriptors: *Genes, Bacterial; * **Staphylococcus** aureus--genetics--GE; *Trans-Activators; Amino Acid Sequence; Bacterial Proteins--genetics--GE; Base Sequence; Cell Division--genetics--GE; Chromosome Mapping; DNA Primers --genetics--GE; DNA, Bacterial--genetics--GE; Genes, Regulator; Molecular Sequence Data; Mutation; Phenotype; Signal Transduction--genetics--GE; **Staphylococcus** aureus--growth and development--GD; **Staphylococcus** aureus--pathogenicity--PY; Virulence--genetics--GE

Molecular Sequence Databank No.: GENBANK/U46541
CAS Registry No.: 0 (Bacterial Proteins); 0 (DNA Primers); 0 (DNA, Bacterial); 0 (SarA protein, bacterial); 0 (Trans-Activators)
Record Date Created: 19960926
Record Date Completed: 19960926

7/9/9 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011179515 BIOSIS NO.: 199799813575
Regulation of virulence factors gene expression in Staphylococcus **aureus**
AUTHOR: Wojcik Kinga Jadwiga
AUTHOR ADDRESS: Al. Mickiewicza 3, 31-120 Krakow, Poland**Poland
JOURNAL: Postepy Biologii Komorki 24 (SUPPL. 8): p77-85 1997 1997
ISSN: 0324-833X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Polish

ABSTRACT: The pathogenesis of **Staphylococcus** aureus which cause a variety of diseases, involves primarily the production and secretion of toxins. The synthesis of virulence factors and other exoproteins by S. aureus is controlled by a global regulatory system agr, and in addition by loci **sar** and xpr which modulate agr response. Expression of secreted proteins is activated in postexponential growth phase, when synthesis of surface proteins is rather inhibited. Locus agr contains two transcription units, driven by **promoters** **P2** and P3. P2 transcript encodes proteins of the classical bacterial two-component transduction system and autocrine factor which activate agr response. P3 operon encodes RNAIII, which is the effector of this system. RNAIII regulates target gene expression at transcriptional level and, by an independent mechanism, stimulates translation of at least one or two of the exoproteins. This described regulatory system probably has analogs in the other bacterial strains. Recognition of mechanisms of virulence genes' expression may help to control bacterial infections by suppression of their virulence.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Genetics; Molecular Genetics--Biochemistry and Molecular Biophysics; Physiology
BIOSYSTEMATIC NAMES: Micrococcaceae--Gram-Positive Cocci, Eubacteria, Bacteria, Microorganisms
ORGANISMS: **Staphylococcus** aureus (Micrococcaceae)
COMMON TAXONOMIC TERMS: Bacteria; Eubacteria; Microorganisms
MISCELLANEOUS TERMS: EXPRESSION; GENOMIC RNA; MOLECULAR GENETICS; TRANSCRIPTION; VIRULENCE FACTORS
CONCEPT CODES:
10062 Biochemistry studies - Nucleic acids, purines and pyrimidines
10300 Replication, transcription, translation
31000 Physiology and biochemistry of bacteria
31500 Genetics of bacteria and viruses
BIOSYSTEMATIC CODES:
07702 Micrococcaceae

7/9/10 (Item 1 from file: 35)
DIALOG(R)File 35:Dissertation Abs Online

01592502 ORDER NO: AAD97-28479

TRANSCRIPTION OF PATHOGENESIS RELATED GENES IN STAPHYLOCOCCUS AUREUS

Author: DEORA, RAJENDAR K.
Degree: PH.D.
Year: 1997
Corporate Source/Institution: UNIVERSITY OF ILLINOIS AT CHICAGO, HEALTH SCIENCES CENTER (0806)
Source: VOLUME 58/04-B OF DISSERTATION ABSTRACTS INTERNATIONAL. PAGE 1669. 124 PAGES
Descriptors: BIOLOGY, MICROBIOLOGY ; BIOLOGY, MOLECULAR
Descriptor Codes: 0410; 0307

In order to study the biochemical basis of transcriptional regulation of toxin gene expression in **Staphylococcus** aureus, DNA dependent RNA polymerase (RNAP) and sigma factors have been purified and characterized. RNAP was purified from exponentially growing cells. SDS-polyacrylamide gel analysis revealed that the most purified preparation of RNAP has the prototype $\alpha\sb2\beta\beta'\sigma$ subunit structure. The σ subunit cross reacted with a polyclonal antibody against $Bacillus\ subtilis\ \sigma\sp{\rm A}$ and co-migrated with the $B.\ subtilis\ \sigma\sp{\rm A}$ subunit. Immunoblot analyses also revealed that the purified RNAP is deficient in σ factor. The purified RNAP was poorly active in transcription assays.

Based on amino acid sequence homology of the B. subtilis vegetative $\sigma\sp{\rm A}$ sigma factor and the predicted product of the chromosomally-located plaC gene of S. aureus, it was hypothesized that plaC could encode the vegetative σ factor. The plaC gene was cloned under the T7 promoter and overexpressed in Escherichia coli strain BL21(DE3)pLysE. The overproduced protein, present in inclusion bodies, was solubilized with guanidine hydrochloride, renatured and purified by DEAE-sephacel and Sephadex G-75 chromatography. The purified protein, designated as $\sigma\sp{\rm SA}$, comigrated with the 55 kDa $\sigma\sp{\rm A}$ subunit and cross-reacted with anti $\sigma\sp{\rm A}$ antibody. E. coli core RNAP, reconstituted with $\sigma\sp{\rm SA}$, initiated promoter-specific transcription from several toxin gene promoters e.g. hla, sea, sec from S. aureus and from the several E. coli $\sigma\sp{70}$-dependent promoters. $\sigma\sp{\rm SA}$, when added to the purified RNAP from S. aureus, stimulated transcriptional activity of the RNAP from the agr **P2** **promoter** by 30-fold and that from the sea promoter by 72-fold. As determined by primer extension studies, the 5$\sp\prime$ ends of the $\sigma\sp{\rm SA}$-initiated mRNAs synthesized in vitro from the agr **P2** and sea **promoters** are in general agreement with the 5$\sp\prime$ ends of the cellular RNAs. Disruption of the plaC gene on the S. aureus chromosome was lethal. plaC encodes the primary σ factor in S. aureus. The purified RNAP reconstituted with $\sigma\sp{\rm SA}$ failed to transcribe from the **sar** P3 promoter, having homology to the $\sigma\sp{\rm B}$-dependent promoters. The putative gene sigB encoding for $\sigma\sp{\rm B}$ of S. aureus was amplified from the chromosome and cloned under the T7 promoter. The overexpressed protein--designated as $\sigma\sp{\rm SB}$-- initiated transcription from the **sar** P3 promoter. Primer extension analyses confirmed that $\sigma\sp{\rm SB}$ directs the RNAP to initiate specific transcription. $\sigma\sp{\rm SB}$ is the first alternate sigma factor from S. aureus. Based on the in vitro transcription data it is speculated that $\sigma\sp{\rm SB}$ plays an important role in modulating the toxin gene regulation.

03255265 H.W. WILSON RECORD NUMBER: BGSI96005265

Characterization of the sar locus and its interaction with agr in Staphylococcus aureus.
Heinrichs, Jon H
Bayer, Manfred G; Cheung, Ambrose L
Journal of Bacteriology (J Bacteriol) v. 178 no2 (Jan. '96) p. 418-23
DOCUMENT TYPE: Feature Article
SPECIAL FEATURES: bibl il ISSN: 0021-9193
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
RECORD TYPE: Abstract RECORD STATUS: New record

ABSTRACT: The interaction between the global regulatory systems **sar** and agr in the expression of cell wall and extracellular proteins in **Staphylococcus** aureus was examined. A DNA fragment situated upstream of **sarA** was found to restore the production of exoproteins by a **sar** mutant to parental levels when the fragment was combined with **sarA** . Cell extracts of the complemented mutant bound to the **P2 promoter** region of the agr locus that controls RNAII transcription. As RNAII promotes the transcription of the agr regulatory molecule, the findings indicate that the **sar** locus may regulate the synthesis of exoprotein by binding to the agr **P2 promoter** region, thereby controlling the agr-mediated pathway of exoprotein production.

DESCRIPTORS:
Bacterial proteins; Genetic regulation--Bacteria; **Staphylococcus**

7/9/12 (Item 1 from file: 266)
DIALOG(R)File 266:FEDRIP

00565843
IDENTIFYING NO.: 5R01AI037142-09 AGENCY CODE: CRISP
CHARACTERIZATION OF SAR /AGR INTERACTIONS IN S AUREUS
PRINCIPAL INVESTIGATOR: CHEUNG, AMBROSE L.
ADDRESS: Ambrose.Cheung@Dartmouth.Edu DARTMOUTH MEDICAL COLLEGE VAIL 206 HANOVER, NH 03755
PERFORMING ORG.: DARTMOUTH COLLEGE, HANOVER, NEW HAMPSHIRE
SPONSORING ORG.: NATIONAL INSTITUTE OF ALLERGY AND INFECTIOUS DISEASES
DATES: 2007/01/96 TO 2005/31/05 FY : 2004 TYPE OF AWARD: Noncompeting Continuation (Type 5)
SUMMARY: Because of increasing antibiotic resistance, **Staphylococcus** aureus continues to be a major human pathogen. To develop a novel approach against this pathogen we have tried to understand the genetic control apparatus in an attempt to identify new targets amenable to therapy. Using Tn917 mutagenesis , we identified a locus on the S. aureus chromosome, designated **sar** , that is involved in the regulation of several extracellular and cell wall virulence factors. The **sar** locus is composed of three overlapping transcripts, each encoding **SarA** , the major **sar** regulatory molecule. The **SarA** protein (14.5 kD) binds to the agr promoter region to modulate transcription of RNAII and RNAIII (the agr regulatory molecule) from the agr **P2** and P3 **promoters** . As agr is a pleiotropic regulator of exoprotein synthesis, our data are consistent with the hypothesis that **SarA** positively regulates the expression of exoprotein genes via agr. The binding site of **SarA** on the agr promoter has been mapped to a 29-bp sequence in the P2-P3 interpromoter region. Sequence alignment revealed a conserved " **SarA** recognition motif" upstream of the -35 promoter boxes of several **sar** target genes (e.g. hla, spa and fnbB) that is homologous with the 29-bp sequence. Deletion of the " **SarA** recognition motif" in the agr and the spa promoter regions renders the

respective genes unresponsive to the effect of the **sar** locus. To verify the hypothesis that **SarA** binds to a conserved **SarA** recognition motif in various target genes to modulate transcription we propose to examine the interactions of **SarA** with target promoters (hla, fnbB and spa) lacking the **SarA** recognition motif. These studies will be followed by footprinting and in vitro transcription assays of target promoters in the presence of **SarA** . These in vitro data will be confirmed by in vivo transcription study of S. aureus cells carrying **sar** target genes lacking the SarA recognition motif. A corollary to our hypothesis is that the activation of **sar** target genes may depend on the **SarA** protein level which, in turn, may be controlled by **SarA** and genetic elements within the extensive 800-bp **sar** promoter region. Additionally, a 13 kD protein, designated **SarR** , may bind to the **sar** promoter region to modulate **sar** transcription and ultimately **SarA** expression. We thus propose to evaluate the contribution of these genetic elements and regulatory proteins in regulating **SarA** expression and hence target gene transcription. The results of these studies will provide a unifying hypothesis for **sar** -mediated regulation whereby **SarA** binds to the conserved **SarA** recognition motif to control target gene transcription and that activation of these promoters is dependent on the **SarA** protein levels. This knowledge is indispensable if we are to design synthetic analogs to interfere with the expression of virulence genes controlled by the **sar** locus in the future.

DESCRIPTORS: **Staphylococcus** aureus; open reading frame; genetic promoter element; genetic transcription; bacterial genetics; gene mutation; antibody; intermolecular interaction; DNA footprinting; bacterial protein; DNA binding protein; gel mobility shift assay

?

13853573 PMID: 11527706

Inhibitors of bacterial enoyl acyl carrier protein reductase (FabI): 2,9-disubstituted 1,2,3,4-tetrahydropyrido[3,4-b]indoles as potential antibacterial agents.

Seefeld M A; Miller W H; Newlander K A; Burgess W J; Payne D J; Rittenhouse S F; Moore T D; DeWolf W E; Keller P M; Qiu X; Janson C A; Vaidya K; Fosberry A P; Smyth M G; Jaworski D D; Slater-Radosti C; Huffman W F

GlaxoSmithKline Pharmaceuticals, Antimicrobial and Host Defense Division, 1250 S. Collegeville Road, Collegeville, PA 19426, USA. mark a seefeld@sbphrd.com

Bioorganic & medicinal chemistry letters (England) Sep 3 2001, 11 (17) p2241-4, ISSN 0960-894X Journal Code: 9107377

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

An **SAR** study of a screening **lead** has led to the identification of 2,9-disubstituted 1,2,3,4-tetrahydropyrido[3,4-b]indoles as inhibitors of **Staphylococcus** aureus enoyl acyl carrier protein reductase (FabI).

Descriptors: *Anti-Bacterial Agents--pharmacology--PD; *Enzyme Inhibitors --chemistry--CH; *Enzyme Inhibitors--pharmacology--PD; *Oxidoreductases --antagonists and inhibitors--AI; Anti-Bacterial Agents--chemistry--CH; Escherichia coli--drug effects--DE; Inhibitory Concentration 50; Microbial Sensitivity Tests; Staphylococcus aureus--drug effects--DE; Structure-Activity Relationship; Triclosan--pharmacology--PD

Molecular Sequence Databank No.: PDB/1I30

CAS Registry No.: 0 (Anti-Bacterial Agents); 0 (Enzyme Inhibitors); 3380-34-5 (Triclosan)

Enzyme No.: EC 1. (Oxidoreductases); EC 1.3.1.9 (enoyl-(acyl-carrier-protein) reductase (NADH))

Record Date Created: 20010830

Record Date Completed: 20020103

13909490 PMID: 11598065

Diminished virulence of an alpha-toxin mutant of Staphylococcus aureus in experimental brain abscesses.

Kielian T; Cheung A; Hickey W F

Department of Pathology, Dartmouth-Hitchcock Medical Center, Dartmouth Medical School, Lebanon, New Hampshire 03756, USA. KielianTammyL@uams.edu

Infection and immunity (United States) Nov 2001, 69 (11) p6902-11, ISSN 0019-9567 Journal Code: 0246127

Contract/Grant No.: NA-27321; NA; NASA; NS40730; NS; NINDS

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Staphylococcus aureus is one of the major etiologic agents of brain abscesses in humans, occasionally **leading** to focal neurological deficits and even death. The objective of the present study was to identify key virulence determinants contributing to the pathogenesis of S. aureus in the brain using a murine brain abscess model. The importance of virulence factor production in disease development was demonstrated by the inability of heat-inactivated S. aureus to induce proinflammatory cytokine or chemokine expression or brain abscess formation in vivo. To directly address the contribution of virulence determinants in brain abscess development, the abilities of S. aureus strains with mutations in the global regulatory loci **sarA** and agr were examined. An S. aureus **sarA** agr double mutant exhibited reduced virulence in vivo, as demonstrated by attenuated proinflammatory cytokine and chemokine expression and bacterial replication. Subsequent studies focused on the expression of factors that are altered in the **sarA** agr double mutant. Evaluation of an alpha-toxin mutant revealed a phenotype similar to that of the **sarA** agr mutant in vivo, as evidenced by lower bacterial burdens and attenuation of cytokine and chemokine expression in the brain. This suggested that alpha-toxin is a central virulence determinant in brain abscess development. Another virulence mechanism utilized by staphylococci is intracellular survival. Cells recovered from brain abscesses were shown to harbor S. aureus intracellularly, providing a means by which the organism may establish chronic infections in the brain. Together, these data identify alpha-toxin as a key virulence determinant for the survival of S. aureus in the brain.

Tags: Male; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Brain Abscess--microbiology--MI; *Phospholipase C --physiology--PH; *Staphylococcal Infections--microbiology--MI; *Staphylococcus aureus--pathogenicity--PY; *Trans-Activators; Animals; Bacterial Proteins--genetics--GE; Bacterial Proteins--physiology--PH; Disease Models, Animal; Heating; Intracellular Fluid--microbiology--MI; Lipase --metabolism--ME; Mice; Mice, Inbred AKR; Phospholipase C--genetics--GE; Staphylococcus aureus--genetics--GE; Tr

11937550 PMID: 9211714

The sae locus of Staphylococcus aureus controls exoprotein synthesis at the transcriptional level.

Giraudo A T; Cheung A L; Nagel R

Departamento de Microbiologia, Facultad de Ciencias Exactas, Fisico-Quimicas y Naturales, Universidad Nacional Rio Cuarto, Ruta 36 Km 601, 5800 Rio Cuarto, Cordoba, Argentina.

Archives of microbiology (GERMANY) Jul 1997, 168 (1) p53-8, ISSN 0302-8933 Journal Code: 0410427

Contract/Grant No.: AI30061; AI; NIAID

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Agr and **sar** are known regulatory loci of **Staphylococcus** aureus that control the production of several extracellular and cell-wall-associated proteins. A pleiotropic insertional mutation in S. aureus, designated sae, that **leads** to the production of drastically diminished levels of alpha- and beta-hemolysins and coagulase and slightly reduced levels of protein A has been described. The study of the expression of the genes coding for these exoproteins in the sae::Tn551 mutant (carried out in this work by Northern blot analyses) revealed that the genes for alpha- and beta-hemolysins (hla and hlb) and coagulase (coa) are not transcribed and that the gene for protein A (spa) is transcribed at a somewhat reduced level. These results indicate that the sae locus regulates these exoprotein genes at the transcriptional level. Northern blot analyses also show that the sae mutation does not affect the expression of agr or **sar** regulatory loci. An sae::Tn551 agr::tetM double mutant has been phenotypically characterized as producing reduced or null levels of alpha-, beta-, and delta-hemolysins, coagulase, and high levels of protein A. Northern blot analyses carried out in this work with the double mutant revealed that hla, hlb, hld, and coa genes are not transcribed, while spa is transcribed at high levels. The fact that coa is not expressed in the sae agr mutant, as in the sae parental strain, while spa is expressed at the high levels characteristic of the agr parental strain, suggests that sae and agr interact in a complex way in the control of the expression of the genes of several exoproteins.

Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Coagulase--metabolism-

The virulence of Staphylococcus aureus is essentially determined by cell wall associated proteins and secreted toxins that are regulated and expressed according to growth phases and/or growth conditions. Gene expression is regulated by specific and sensitive mechanisms, most of which act at the transcriptional level. Regulatory factors constitute numerous complex networks, driving specific interactions with target gene promoters. These factors are largely regulated by two-component regulatory systems, such as the agr, saeRS, srrAB, arlSR and lytRS systems. These systems are sensitive to environmental signals and consist of a sensor histidine kinase and a response regulator protein. DNA-binding proteins, such as SarA and the recently identified SarA homologues (**SarR** , Rot, SarS, SarT, SarU), also regulate virulence factor expression. These homologues might be intermediates in the regulatory networks. The multiple pathways generated by these factors allow the bacterium to adapt to environmental conditions rapidly and specifically, and to develop infection. Precise knowledge of these regulatory mechanisms and how they control virulence factor expression would open up new perspectives for antimicrobial chemotherapy using key inhibitors of these systems. (114 Refs.)

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Gene Expression Regulation, Bacterial; *Staphylococcus aureus--genetics--GE; *Staphylococcus aureus--pathogenicity--PY; *Virulence Factors--genetics--GE; Amino Acid Sequence; Molecular Sequence Data; Virulence

CAS Registry No.: 0 (Virulence Factors)

Record Date Created: 20040427

Record Date Completed: 20040702

4/9/3

DIALOG(R)File 155:MEDLINE(R)

14860092 PMID: 12837797

Crystal structure of the SarS protein from Staphylococcus aureus.

Li Ronggui; Manna Adhar C; Dai Shaodong; Cheung Ambrose L; Zhang Gongyi

Integrated Department of Immunology, National Jewish Medical and Research Center, and Department of Pharmacology, Biomolecular Structure Program, School of Medicine, University of Colorado Health Science Center, Denver, Colorado 80206, USA.

Journal of bacteriology (United States) Jul 2003, 185 (14) p4219-25, ISSN 0021-9193 Journal Code: 2985120R

Contract/Grant No.: AI50678; AI; NIAID

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

The expression of virulence determinants in Staphylococcus aureus is controlled by global regulatory loci (e.g., sarA and agr). One of these determinants, protein A (spa), is activated by sarS, which encodes a 250-residue DNA-binding protein. Genetic analysis indicated that the agr locus likely mediates spa repression by suppressing the transcription of sarS. Contrary to SarA and **SarR** , which require homodimer formation for proper function, SarS is unusual within the SarA protein family in that it contains two homologous halves, with each half sharing sequence similarity to SarA and **SarR** . Here we report the 2.2 A resolution X-ray crystal structure of the SarS protein. SarS has folds similar to those of **SarR** and, quite plausibly, the native SarA structure. Two typical winged-helix DNA-binding domains are connected by a well-ordered loop. The interactions between the two domains are extensive and conserved. The putative

DNA-binding surface is highly positively charged. In contrast, negatively charged patches are located opposite to the DNA-binding surface. Furthermore, sequence alignment and structural comparison revealed that MarR has folds similar to those of **SarR** and SarS. Members of the MarR protein family have previously been implicated in the negative regulation of an efflux pump involved in multiple antibiotic resistance in many gram-negative species. We propose that MarR also belongs to the winged-helix protein family and has a similar mode of DNA binding as **SarR** and SarS and possibly the entire SarA protein family member. Based on the structural differences of **SarR** , SarS, and MarR, we further classified these winged-helix proteins to three subfamilies, SarA, SarS, and MarR. Finally, a possible transcription regulation mechanism is proposed.

Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Bacterial Proteins--chemistry--CH; *DNA-Binding Proteins --chemistry--CH; *Staphylococcus aureus--metabolism--ME; Bacterial Proteins --genetics--GE; Bacterial Proteins--metabolism--ME; Crystallization; Crystallography, X-Ray; DNA, Bacterial--chemistry--CH; DNA, Bacterial --metabolism--ME; DNA-Binding Proteins--genetics--GE; DNA-Binding Proteins --metabolism--ME; Gene Expression Regulation, Bacterial; Humans; Hydrophobicity; Models, Molecular; Protein Conformation; Protein Structure, Secondary; Protein Structure, Tertiary; Staphylococcus aureus--chemistry --CH; Staphylococcus aureus--genetics--GE; Transcription Factors --chemistry--CH; Transcription Factors--genetics--GE; Transcription Factors--metabolism--ME

Molecular Sequence Databank No.: PDB/1P4X

CAS Registry No.: 0 (Bacterial Proteins); 0 (DNA, Bacterial); 0 (DNA-Binding Proteins); 0 (SarS protein, Staphylococcus aureus); 0 (Transcription Factors)

Record Date Created: 20030702

Record Date Completed: 20030808

4/9/4

DIALOG(R)File 155:MEDLINE(R)

14316394 PMID: 12133812

Global regulation of virulence determinants in Staphylococcus aureus by the SarA protein family.

Cheung Ambrose L; Zhang Gongyi

Department of Microbiology and Immunology, Dartmouth Medical School, Hanover, NH 03755, USA. ambrose.cheung@dartmouth.edu

Frontiers in bioscience - a journal and virtual library (United States) Aug 1 2002, 7 pd1825-42, ISSN 1093-4715 Journal Code: 9709506

Contract/Grant No.: AI37142; AI; NIAID; AI50678; AI; NIAID

Publishing Model Electronic

Document type: Journal Article; Review; Review, Tutorial

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

In S. aureus, the production of virulence determinants such as cell wall adhesins and exotoxins during the growth cycle is controlled by global regulators such as SarA and agr. Genomic scan reveals 16 two-component regulatory systems (e.g. agr and sae) as well as a family of SarA homologs in S. aureus. We call the SarA homologs the SarA protein family. Many of the members in this protein family are either small basic proteins (<153 residues) or two-domain proteins in which a single domain shares sequence similarity to each of the small basic proteins. Recent crystal structures of **SarR** and SarA reveal dimeric structures for these proteins. Because of

its structure and unique mode of DNA binding, **SarR** , and possibly other SarA family members, may belong to a new functional class of the winged-helix family, accommodating long stretch of DNA with bending points. Based on sequence homology, we hypothesize that the SarA protein family may entail homologous structures with similar DNA-binding motifs but divergent activation domains. An understanding of how these regulators interact with each other in vivo and how they sense environmental signals to control virulence gene expression (e.g. alpha-hemolysin) will be important to our eventual goal of disrupting the regulatory network. (122 Refs.)

Tags: Comparative Study; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Bacterial Proteins--physiology--PH; *Gene Expression Regulation, Bacterial--physiology--PH; *Staphylococcus aureus --pathogenicity--PY; *Trans-Activators--physiology--PH; Amino Acid Sequence ; Bacterial Proteins--chemistry--CH; Bacterial Proteins--genetics--GE; Gene Expression Regulation, Bacterial--genetics--GE; Molecular Sequence Data; Multigene Family--genetics--GE; Multigene Family--physiology--PH; Sequence Homology, Amino Acid; Staphylococcus aureus--genetics--GE; Trans-Activators--chemistry--CH; Trans-Activators--genetics--GE; Virulence --genetics--GE

CAS Registry No.: 0 (Bacterial Proteins); 0 (SarA protein, bacterial) ; 0 (Trans-Activators)

Record Date Created: 20020722

Record Date Completed: 20021010

Date of Electronic Publication: 20020801

4/9/5

DIALOG(R)File 155:MEDLINE(R)

14278658 PMID: 12086893

Introduction of Dr. Michael Sarr .

Beger H G

University of Ulm, Germany.

Journal of gastrointestinal surgery - official journal of the Society for Surgery of the Alimentary Tract (United States) Nov-Dec 2001, 5 (6) p569-71, ISSN 1091-255X Journal Code: 9706084

Publishing Model Print

Document type: Addresses; Biography; Historical Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

Descriptors: *Surgery--history--HI; History, 20th Century; Leadership; Portraits; Societies, Medical--organization and administration--OG; United States

Named Person: **Sarr** M

Record Date Created: 20020627

Record Date Completed: 20020822

4/9/6

DIALOG(R)File 155:MEDLINE(R)

14244378 PMID: 12049370

Arrhythmic risk stratification after myocardial infarction using ambulatory electrocardiography signal averaging.

Roche Frederic; DaCosta Antoine; Karnib Ibrahim; Triomphe Geraldine; Roche Christian; Isaaz Karl; Geyssant Andre; Barthelemy Jean-Claude

--genetics--GE; Staphylococcus aureus--pathogenicity--PY; Virulence
CAS Registry No.: 0 (Bacterial Proteins); 0 (SarA protein, bacterial)
; 0 (Trans-Activators)
Record Date Created: 20011130
Record Date Completed: 20020211

4/9/8
DIALOG(R)File 155:MEDLINE(R)

13783452 PMID: 11447147
SarT, a repressor of alpha-hemolysin in Staphylococcus aureus.
Schmidt K A; Manna A C; Gill S; Cheung A L
Department of Microbiology, Dartmouth Medical School, Hanover, New Hampshire 13755, USA. Katherine.a.schmidt@dartmouth.edu
Infection and immunity (United States) Aug 2001, 69 (8) p4749-58, ISSN 0019-9567 Journal Code: 0246127
Contract/Grant No.: A107519-14; PHS; AI37142; AI; NIAID; AI43968; AI; NIAID
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS
In searching the Staphylococcus aureus genome, we found several homologs to SarA. One of these genes, sarT, codes for a basic protein with 118 residues and a predicted molecular size of 16,096 Da. Northern blot analysis revealed that the expression of sarT was repressed by sarA and agr. An insertion sarT mutant generated in S. aureus RN6390 and 8325-4 backgrounds revealed minimal effect on the expression of **sarR** and sarA. The RNAIII level was notably increased in the sarT mutant, particularly in postexponential-phase cells, while the augmentative effect on RNAII was less. SarT repressed the expression of alpha-hemolysin, as determined by Northern blotting, Western blotting, and a rabbit erythrocyte hemolytic assay. This repression was relieved upon complementation. Similar to agr and sarA mutants, which predictably displayed a reduction in hla expression, the agr sarT mutant exhibited a lower level of hla transcription than the sarT mutant. In contrast, hla transcription was enhanced in the sarA sarT mutant compared with the single sarA mutant. Collectively, these results indicated that the sarA locus, contrary to the regulatory action of agr, induced alpha-hemolysin production by repressing sarT, a repressor of hla transcription.
Tags: Research Support, U.S. Gov't, P.H.S.
Descriptors: *Bacterial Proteins--genetics--GE; *Bacterial Toxins --genetics--GE; *Gene Expression Regulation, Bacterial; *Hemolysins --genetics--GE; *Repressor Proteins--genetics--GE; *Staphylococcus aureus --genetics--GE; *Trans-Activators; Amino Acid Sequence; Animals; Bacterial Proteins--metabolism--ME; Bacterial Toxins--biosynthesis--BI; Base Sequence ; Blotting, Western--methods--MT; DNA, Bacterial; Hemolysins--biosynthesis --BI; Molecular Sequence Data; Mutagenesis; Phenotype; Rabbits; Repressor Proteins--metabolism--ME; Staphylococcus aureus--metabolism--ME; Transcript ion Factors
CAS Registry No.: 0 (Bacterial Proteins); 0 (Bacterial Toxins); 0 (DNA, Bacterial); 0 (Hemolysins); 0 (Repressor Proteins); 0 (SarA protein, bacterial); 0 (SarH1 protein, Staphylococcus aureus); 0 (SarH3 protein, Staphylococcus aureus); 0 (Trans-Activators); 0 (Transcription Factors); 0 (staphylococcal alpha-toxin)
Record Date Created: 20010711
Record Date Completed: 20010823

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          143712  S2
      S3      12  S1 AND S2
? t s3/9/all
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ISSN 1350-4533 Journal Code: 9422753
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS

This study investigates the optimal external parameters for using an ultrasound applicator for treating bone tumors. This system utilized spherically arranged applicators such as scanned focused ultrasound, and spherically focused multielement applicators. The power deposition pattern is modeled as geometric gain with exponential attenuation. The specific absorption rate ratio (**SARR**) criteria have been used to determine the proper heating domain of ultrasound driving frequency and therapeutic tumor diameter. The results demonstrate that the optimal driving frequency depends on tumor depth, ultrasound absorption of bone marrow, and diameter of bone, but it is independent of the acoustic window area and **SARR** . The treatable diameter of bone tumor increased when the absorption ratio of bone marrow to tumor, acoustic window of surface skin, and diameter of bone were elevated. However, the treatable diameter of bone tumor decreased when muscle thickness, **SARR** of bone tumor site to the surface skin, bone marrow, and bone declined. To deliver the ultrasound energy into the tumor site and to avoid the potential damage to the normal tissue as much as possible, the specific absorption rate (SAR) in the bone tumor site has to be three times higher than that in the surface skin, tumor/marrow, and marrow/bone interfaces. The temperature distributions can verify the **SARR** criteria in this model. This study provides the information for choosing the optimal operating frequency of the ultrasound transducer and the acoustic window on the skin surface, and for designing the ultrasound applicator for clinical implementation.

Tags: Research Support, Non-U.S. Gov't

Descriptors: *Bone Neoplasms--therapy--TH; *Ultrasonic Therapy; Biomedical Engineering; Bone Marrow--pathology--PA; Bone Neoplasms--blood supply--BS; Bone Neoplasms--pathology--PA; Humans; Models, Theoretical; Temperature; Ultrasonic Therapy--instrumentation--IS; Ultrasonic Therapy --methods--MT

Record Date Created: 20001211
Record Date Completed: 20001211

4/9/12
DIALOG(R)File 155:MEDLINE(R)

08413813 PMID: 3191498

Personal and family history of lung disease as risk factors for adenocarcinoma of the lung.

Wu A H; Yu M C; Thomas D C; Pike M C; Henderson B E
University of Southern California, Los Angeles 90033.

Cancer research (UNITED STATES) Dec 15 1988, 48 (24 Pt 1) p7279-84,
ISSN 0008-5472 Journal Code: 2984705R
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Subfile: INDEX MEDICUS

To identify risk factors for adenocarcinoma of the lung, a population-based case-control study of 336 female cancers of this cell type and an equal number of neighborhood controls was conducted between 1983 and 1986. After adjusting for personal smoking, personal and family histories

of lung disease emerged as additional risk factors. A personal history of any lung disease was associated with a 40% increase in risk [smoking adjusted relative risk (**SARR**) = 1.4, 95% confidence interval (CI) = 1.0, 2.0] with a more marked increase in risk for lung diseases occurring during childhood (**SARR** = 1.9, 95% CI = 1.2, 3.2) and for tuberculosis (**SARR** = 10.0, 95% CI = 1.1, 90.1). Family histories of tuberculosis (**SARR** = 2.0, 95% CI = 1.1, 3.6) and of lung cancer (**SARR** = 3.9, 95% CI = 2.0, 7.6) were also risk factors for adenocarcinoma of the lung. Increasing risk was observed with decreasing intake of dietary beta-carotene. After adjusting for personal smoking, women in the lowest quartile of intake showed a two-fold increased risk relative to those in the highest quartile of intake (P = 0.003). There were also some suggestive differences between cases and controls in their reproductive history and hormone use.

Tags: Female; Research Support, Non-U.S. Gov't

Descriptors: *Adenocarcinoma--etiology--ET; *Lung Diseases--etiology--ET; *Lung Diseases--genetics--GE; Adult; Humans; Middle Aged; Risk Factors; Smoking

Record Date Created: 19890109

Record Date Completed: 19890109

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Logoff: level 05.06.01 D 16:04:19

You are now logged off

DERWENT-ACC-NO: 2002-706985
DERWENT-WEEK: 200433

TITLE: New sarR gene from the locus of Staphylococcus aureus, useful for treating gram-positive bacteremia

INVENTOR: CHEUNG, A L; MANNA, A ; ZHANG, G

PATENT-ASSIGNEE: DARTMOUTH COLLEGE (DARTN), CHEUNG A L (CHEUI), MANNA A (MANNI), ZHANG G (ZHANI)

PRIORITY-DATA: 2001US-289601P (May 8, 2001), 2001US-261233P (January 12, 2001), 2001US-261607P (January 12, 2001), 2002US-0043539 (January 11, 2002)

Search Selected | Search ALL | Clear

PATENT-FAMILY:

	PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
☐	AU 2002243519 A1	September 12, 2002		000	C12N000/00
☐	WO 200268610 A2	September 6, 2002	E	062	C12N000/00
☐	US 20030114650 A1	June 19, 2003		000	C07H021/02

DESIGNATED-STATES: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZM ZW AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
AU2002243519A1	January 11, 2002	2002AU-0243519	
AU2002243519A1		WO 200268610	Based on
WO 200268610A2	January 11, 2002	2002WO-US00877	
US20030114650A1	January 12, 2001	2001US-261233P	Provisional
US20030114650A1	January 12, 2001	2001US-261607P	Provisional
US20030114650A1	May 8, 2001	2001US-289601P	Provisional
US20030114650A1	January 11, 2002	2002US-0043539	

INT-CL (IPC): C07 H 21/02; C07 H 21/04; C12 N 0/00

ABSTRACTED-PUB-NO: WO 200268610A
BASIC-ABSTRACT:

NOVELTY - An isolated nucleic acid sequence (I), which regulates the expression of virulence determinants in gram-positive bacteria, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) a vector (II) comprising the nucleic acid sequence;

(2) a host cell (III) comprising the vector;

(3) a method (IV) for identifying putative agents that inhibit growth and infectivity of bacteria;

(4) a method (V) of inhibiting growth and infectivity of bacteria;

(5) a pharmaceutical composition (VI) for use as an anti-bacterial agent comprising the agent that enhances the expression of the nucleic acid sequence or the activity of the polypeptide that it encodes and a vehicle or the compound identified by the method of (X) or a compound that binds to the P1 promoter region of a sarA gene;

(6) an isolated polypeptide (VII) that regulates the expression of virulence determinants in gram-positive bacteria;

(7) a kit (VIII) for identifying the presence of a sarR gene or its product comprising a means for analyzing a biological sample for the presence of the sarR gene or its product;

(8) a method (IX) of treating a mammal suffering from or susceptible to a gram-positive bacterial infection; or

(9) a method (X) of screening for lead compounds that inhibit the expression of virulence determinants in gram-positive bacteria.

ACTIVITY - Antibacterial.

No biological data given.

MECHANISM OF ACTION - SarR-Agonist.

USE - The pharmaceutical composition comprising a sarR agonist or a compound capable of selective occupation of a sarA promoter receptor is useful for treating gram-positive bacteremia (claimed).

ABSTRACTED-PUB-NO: WO 200268610A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/12

DERWENT-CLASS: B04 D16
CPI-CODES: B04-C01; B04-E01; B04-E02F; B04-E03F; B04-E08; B04-F0100E; B04-F10B; B04-F10B0E; B04-F10B3; B04-F10B3E; B04-N03A0E; B11-A01; B11-C08E1; B11-C08F; B11-C08F2; B11-C08F4; B11-C08G; B11-C10; B12-K04E; B14-A01B; B14-A01B4; D05-H04; D05-H08; D05-H09; D05-H12A; D05-H12E; D05-H14; D05-H17A6; D05-H18;

Previous Doc Next Doc Go to Doc#

*File 467: F467 no longer updates; see Help News467. 7.

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 8/9/11   (Item 7 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)

0012434986   BIOSIS NO.: 200000153299
A winged helix protein from yeast Saccharomyces cerevisiae recognizes centromere sequences
AUTHOR: Myrich Emily; Shiyanova Tatiyana; Liao Xiubei (Reprint)
AUTHOR ADDRESS: Department of Biochemistry and Molecular Biology, College of Medicine, University of Illinois at Chicago, 1819 West Polk Street, M/C 536, Chicago, IL, 60612, USA**USA
JOURNAL: Archives of Biochemistry and Biophysics 375 (1): p78-82 March 1, 2000 2000
MEDIUM: print
ISSN: 0003-9861
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The winged helix-turn-helix motif was initially identified in the mammalian hepatocyte-enriched transcription factor HNF-3 and the Drosophila forkhead homeotic protein. Proteins containing the winged helix motif have been shown to play important roles in tissue-specific developmental **regulation** . In this report, by using a genomic binding site selection method, we demonstrate that the winged helix protein YFKH-1 from the yeast Saccharomyces cerevisiae recognizes conserved sequence in yeast centromeres. Thus, our data suggest that the winged helix proteins of the yeast may be involved in centromeric functions of the yeast.

DESCRIPTORS:
  MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Molecular Genetics --Biochemistry and Molecular Biophysics
  BIOSYSTEMATIC NAMES: Ascomycetes--Fungi, Plantae
  ORGANISMS: Saccharomyces cerevisiae (Ascomycetes)
  ORGANISMS: PARTS ETC: centromere
  COMMON TAXONOMIC TERMS: Fungi; Microorganisms; Nonvascular Plants; Plants
  CHEMICALS & BIOCHEMICALS: HNF-3--hepatocyte-enriched transcription factor; YFKH-1-- **winged helix protein** ; forkhead homeotic protein--Drosophila; **winged helix-turn-helix motif**
  MISCELLANEOUS TERMS: nucleotide sequence--DNA sequence
CONCEPT CODES:
  10060 Biochemistry studies - General
  03504 Genetics - Plant
BIOSYSTEMATIC CODES:
  15100 Ascomycetes

 8/9/40   (Item 6 from file: 34)

07556067 Genuine Article#: 180KN Number of References: 51
Title: **The high-resolution crystal structure of the molybdate-dependent transcriptional regulator (ModE) from Escherichia coli: a novel combination of domain folds**
Author(s): Hall DR; Gourley DG; Leonard GA; Duke EMH; Anderson LA; Boxer DH ; Hunter WN (REPRINT)
Corporate Source: UNIV DUNDEE,DEPT BIOCHEM, WELLCOME TRUST BLDG/DUNDEE DD1 5EH//SCOTLAND/ (REPRINT); UNIV DUNDEE,DEPT BIOCHEM/DUNDEE DD1 5EH//SCOTLAND/; SERC,DARESBURY LAB, CCLR DL/WARRINGTON WA4 4AD/CHESHIRE/ENGLAND/; UNIV DUNDEE,DEPT BIOCHEM/DUNDEE DD1 5HN//SCOTLAND/; EUROPEAN SYNCHROTRON RADIAT FACIL,JOINT STRUCT BIOL GRP/F-38043 GRENOBLE//FRANCE/
Journal: EMBO JOURNAL, 1999, V18, N6 (MAR 15), P1435-1446
ISSN: 0261-4189 Publication date: 19990315
Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND
Language: English Document Type: ARTICLE
Geographic Location: SCOTLAND; ENGLAND; FRANCE
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY; CELL BIOLOGY
Abstract: The molybdate-dependent transcriptional **regulator** (ModE) from Escherichia call functions as a sensor of molybdate concentration and a **regulator** for transcription of operons involved in the uptake and utilization of the essential element, molybdenum. We have determined the structure of ModE using multi-wavelength anomalous dispersion. Selenomethionyl and native ModE models are refined to 1.75 and 2.1 Angstrom, respectively and describe the architecture and structural detail of a complete transcriptional **regulator** . ModE is a homodimer and each subunit comprises N- and C-terminal domains. The N-terminal domain carries a winged helix-turn-helix motif for binding to DNA and is primarily responsible for ModE dimerization. The C-terminal domain contains the molybdate-binding site and residues implicated in binding the oxyanion are identified. This domain is divided into sub-domains a and b which have similar folds, although the organization of secondary structure elements varies. The sub-domain fold is related to the oligomer binding-fold and similar to that of the subunits of several toxins which are involved in extensive protein-protein interactions. This suggests a role for the C-terminal domain in the formation of the ModE-protein-DNA complexes necessary to **regulate** transcription. Modelling of ModE interacting with DNA suggests that a large distortion of DNA is not necessary for complex formation.
Descriptors--Author Keywords: DNA binding ; gene **regulation** ; molybdate ; OB-fold ; **winged helix-turn-helix**
Identifiers--KeyWord Plus(R): BINDING-PROTEIN MOP; CAP-DNA COMPLEX; CLOSTRIDIUM-PASTEURIANUM; TRANSPORT OPERON; PERTUSSIS TOXIN; LYSR FAMILY; MODABCD; SITES; REFINEMENT; REPRESSOR
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MERRITT EA, 1994, V50, P869, ACTA CRYSTALLOGR D
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8/9/45 (Item 1 from file: 71)

01262231 1999243617

C-terminal DNA binding stimulates N-terminal phosphorylation of the outer membrane protein regulator OmpR from Escherichia coli

Ames S.K.; Frankema N.; Kenney L.J.
ADDRESS: L.J. Kenney, Dept. of Molec. Microbiol./Immunol., L-220 Oregon Hlth. Sci. University, 3181 Southwest Sam Jackson Park Road, Portland, OR 97201-3098, United States
EMAIL: kenneyl@ohsu.edu

Journal: Proceedings of the National Academy of Sciences of the United States of America, 96/21 (11792-11797), 1999, United States
CODEN: PNASA
ISSN: 0027-8424
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 56

Expression of the porin genes of Escherichia coli is **regulated** in part by the osmolarity of the growth medium. The process is controlled by the histidine kinase EnvZ and the response **regulator** OmpR. We have previously shown that phosphorylation of OmpR increases its affinity for the upstream **regulatory** regions of ompF and ompC. We now report that, in the presence of DNA, there is a dramatic stimulation in the level of phospho-OmpR. This effect is independent of the source of phosphorylation, i.e., stimulation of phosphorylation is observed with a small phosphorylating agent such as acetyl phosphate or with protein-catalyzed phosphorylation by the kinase EnvZ. The dephosphorylation rate of phospho-OmpR is affected only slightly by the presence of DNA; thus, the increased level is largely caused by an increased rate of phosphorylation. Stimulation of phosphorylation requires specific binding of DNA by OmpR. Occupancy of the DNA binding domain exposes a trypsin cleavage site in the linker, which connects the phosphorylation domain with the DNA binding domain. Our results indicate that when DNA binds in the C terminus, it enhances phosphorylation in the N terminus, and the linker undergoes a conformational change. A generalized mechanism involving a four- state model for response **regulators** is proposed.

DESCRIPTORS:
Winged helix-turn-helix ; Response **regulator** ; Porin **regulation** ; Two-component **regulatory** system; Transcriptional activator

SPECIES DESCRIPTORS:
Escherichia coli
CLASSIFICATION CODE AND DESCRIPTION:
85.1.3.2 - APPLIED MICROBIOLOGY AND BIOTECHNOLOGY / BIOTECHNOLOGY - TECHNIQUES AND PROCEDURES / Culture Selection and Improvement / Recombinant DNA technology
85.7.17.5 - APPLIED MICROBIOLOGY AND BIOTECHNOLOGY / MICROBIAL METABOLISM AND PHYSIOLOGY / Microbial Physiology / Membranes

8/9/31 (Item 27 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010770635 BIOSIS NO.: 199799404695
The DNA-binding domain of OmpR: Crystal structure of a winged helix transcription factor
AUTHOR: Martinez-Hackert Erik; Stock Ann M (Reprint)
AUTHOR ADDRESS: Howard Hughes Med. Inst., Piscataway, NJ 08854, USA**USA
JOURNAL: Structure (London) 5 (1): p109-124 1997 1997
ISSN: 0969-2126
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background: The differential expression of the ompF and ompC genes is **regulated** by two proteins that belong to the two component family of signal transduction proteins: the histidine kinase, EnvZ, and the response **regulator** , OmpR. OmpR belongs to a subfamily of at least 50 response **regulators** with homologous C-terminal DNA-binding domains of 'approximately 98 amino acids. Sequence homology with DNA-binding proteins of known structure cannot be detected, and the lack of structural information has prevented understanding of many of this family's functional properties. Results: We have determined the crystal structure of the Escherichia coli OmpR C-terminal domain at 1.95 ANG

resolution. The structure consists of three alpha helices packed against two antiparallel beta sheets. Two helices, alpha-2 and alpha-3, and the ten residue loop connecting them constitute a variation of the helix-turn-helix (HTH) motif. Helix alpha-3 and the loop connecting the two C-terminal beta strands, beta-6 and beta-7, are probable DNA-recognition sites. Previous mutagenesis studies indicate that the large loop connecting helices alpha-2 and alpha-3 is the site of interaction with the a subunit of RNA polymerase. Conclusions: OmpRc belongs to the family of 'winged helix-turn-helix' DNA-binding proteins. This relationship, and the results from numerous published mutagenesis studies, have helped us to interpret the functions of mos of the structural elements present in this protein domain. The structure of OmpRc could be useful in helping to define the positioning of the alpha subunit of RNA polymerase in relation to transcriptional activators that are bound to DNA.

DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics
BIOSYSTEMATIC NAMES: Enterobacteriaceae--Facultatively Anaerobic Gram-Negative Rods, Eubacteria, Bacteria, Microorganisms
ORGANISMS: Escherichia coli (Enterobacteriaceae)
COMMON TAXONOMIC TERMS: Bacteria; Eubacteria; Microorganisms
MISCELLANEOUS TERMS: ALPHA-HELICES; BETA-SHEETS; BIOCHEMISTRY AND BIOPHYSICS; CRYSTAL STRUCTURE; DNA; DNA-BINDING DOMAIN; DNA-RECOGNITION SITES; HELIX-TURN-HELIX MOTIF; OMPR; PROTEIN-PROTEIN INTERACTION SITE; RNA POLYMERASE ALPHA-SUBUNIT; **WINGED HELIX TRANSCRIPTION FACTOR**
CONCEPT CODES:
10060 Biochemistry studies - General
10502 Biophysics - General
BIOSYSTEMATIC CODES:
06702 Enterobacteriaceae

8/9/32 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010727736 BIOSIS NO.: 199799361796
Aberrant cell growth induced by avian winged helix proteins
AUTHOR: Freyaldenhoven Bettina S (Reprint); Freyaldenhoven Markus P; Iacovoni Jason S; Vogt Peter K
AUTHOR ADDRESS: Scripps Res. Inst., Div. Oncovirol., BCC239, Dep. Mol. Exp. Med., 10550 N. Torrey Pines Rd., La Jolla, CA 92037, USA**USA
JOURNAL: Cancer Research 57 (1): p123-129 1997 1997
ISSN: 0008-5472
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Winged helix transcription factors act as important **regulators** of embryonal development and tissue differentiation in vertebrates and invertebrates. Identification of the retroviral oncogene v-qin as a member of the winged helix family showed that these developmental **regulators** also have oncogenic potential. We used low-stringency hybridization of a chicken embryonic cDNA library to isolate cDNA clones coding for three chicken winged helix (CWH) proteins, CWH-1, CWH-2, and CWH-3. The CWH genes are transcribed in a tissue-restricted pattern in adult and embryonic chicken tissues. The CWH proteins bind to conserved DNA binding sites for winged helix proteins in a sequence-specific manner. Expression of the CWH proteins from replication-competent retroviral RCAS vectors induces changes in morphology and growth pattern

of chicken embryo fibroblasts. CWH-1 and CWH-3 also induce anchorage-independent growth in agar. Chicken embryo fibroblasts expressing the RCAS constructs release replication-competent viruses that are able to elicit the same cellular changes as the parental plasmid DNA. Our results suggest that winged helix transcription factors not only function as **regulators** of development and differentiation but also have the potential to stimulate abnormal cell proliferation.

DESCRIPTORS:
MAJOR CONCEPTS: Cell Biology; Development; Genetics; Molecular Genetics--Biochemistry and Molecular Biophysics; Tumor Biology
BIOSYSTEMATIC NAMES: Galliformes--Aves, Vertebrata, Chordata, Animalia
ORGANISMS: chicken (Galliformes)
COMMON TAXONOMIC TERMS: Animals; Birds; Chordates; Nonhuman Vertebrates; Vertebrates
MISCELLANEOUS TERMS: ADULT; CELL DIFFERENTIATION; CELL PROLIFERATION; DEVELOPMENT; EMBRYO; FIBROBLAST; GENE TRANSCRIPTION; GROWTH PATTERN; MORPHOLOGY; ONCOGENESIS; V-QIN ONCOGENE; **WINGED HELIX TRANSCRIPTION FACTOR**
CONCEPT CODES:
02506 Cytology - Animal
03506 Genetics - Animal
10300 Replication, transcription, translation
24007 Neoplasms - Carcinogens and carcinogenesis
25502 Development and Embryology - General and descriptive
25508 Development and Embryology - Morphogenesis
BIOSYSTEMATIC CODES:
85536 Galliformes

8/9/33 (Item 29 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010596848 BIOSIS NO.: 199699230908
Genesis, a winged helix transcriptional repressor with expression restricted to embryonic stem cells
AUTHOR: Sutton Jill; Costa Robert; Klug Michael; Field Loren; Xu Dawei; Largaespada David A; Fletcher Colin F; Jenkins Nancy A; Copeland Neal G; Klemsz Michael; Hormas Robert (Reprint)
AUTHOR ADDRESS: Div. Hematol./Oncol. Walther Oncol. Cent., IB 442, Indiana Univ. Med. Cent., 975 W. Walnut St., Indianapolis, IN 46202-5121, USA** USA
JOURNAL: Journal of Biological Chemistry 271 (38): p23126-23133 1996 1996
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A novel member of the winged helix (formerly HNF-3/Forkhead) transcriptional **regulatory** family, termed Genesis, was isolated and characterized. Putative translation of the complete cDNA revealed the winged helix DNA binding domain to be centrally located within the protein, with regions on either side that contain known transcriptional **regulatory** motifs. Extensive Northern analysis of Genesis found that the message was exclusively expressed in embryonic stem cells or their malignant equivalent, embryonal carcinoma cells. The Genesis transcript was down- **regulated** when these cells were stimulated to differentiate. DNA sequences that Genesis protein would interact with were characterized and were found to contain a consensus similar to that found in an

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8/9/37 (Item 3 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08588437 Genuine Article#: 304NU Number of References: 52
Title: Forkhead genes in transcriptional silencing, cell morphology and the cell cycle: Overlapping and distinct functions for FKH1 and FKH2 in Saccharomyces cerevisiae
Author(s): Hollenhorst PC; Bose ME; Mielke MR; Muller U; Fox CA (REPRINT)
Corporate Source: UNIV WISCONSIN,DEPT BIOMOL CHEM, 587 MSC, 1300 UNIV AVE/MADISON//WI/53706 (REPRINT); UNIV WISCONSIN,DEPT BIOMOL CHEM/MADISON//WI/53706; UNIV WISCONSIN,DEPT GENET/MADISON//WI/53706
Journal: GENETICS, 2000, V154, N4 (APR), P1533-1548
ISSN: 0016-6731 Publication date: 20000400

Publisher: GENETICS, 428 EAST PRESTON ST, BALTIMORE, MD 21202
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences; CC AGRI--Current Contents, Agriculture, Biology & Environmental Sciences
Journal Subject Category: GENETICS & HEREDITY
Abstract: The SIR1 gene is one of four specialized genes in Saccharomyces cerevisiae required for repressing transcription at the silent mating-type cassettes, HML alpha and HMRa, by a mechanism known as silencing. Silencing requires the assembly of a specialized chromatin structure analogous to heterochromatin. FKH1 was isolated as a gene that, when expressed in multiple copies, could substitute for the function of SIR1 in silencing HMRa. FKH1 (Forkhead Homologue One) was named for its homology to the forkhead family of eukalyotic transcription factors classified on the basis of a conserved DNA binding domain. Deletion of FKH1 caused a defect in silencing HMRa, indicating that FKH1 has a positive role in silencing. Significantly, deletion of both FKH1 and its closest homologue in yeast, FKH2, caused a form of least pseudohyphal growth, indicating that the two genes have redundant functions in controlling yeast cell morphology. By several criteria, fkh1 Delta fkh2 Delta-induced pseudohyphal growth was distinct from the nutritionally induced form of pseudohyphal growth observed in some strains of S. cerevisiae. Although FKH2 is redundant with FKH1 in controlling pseudohyphal growth, the two genes have different functions in silencing HMRa. High-copy expression of CLB2, a G2/M-phase cyclin, prevented fkh1 Delta fkh2 Delta-induced pseudohyphal growth and **modulated** some of the fkh Delta-induced silencing phenotypes. Interestingly, deletions in either FKH1 or FKH2 alone caused subtle but opposite effects on cell-cycle progression and CLB2 mRNA expression, consistent with a role for each of these genes in **modulating** the cell cycle and having opposing effects on silencing. The differences between Fkh1p and Fkh2p in vivo were not attributable to differences in their DNA binding domains.
Identifiers--KeyWord Plus(R): ORIGIN RECOGNITION COMPLEX; POLYMERASE CHAIN-REACTION; DNA-BINDING SPECIFICITY; **WINGED HELIX PROTEINS**; TELOMERIC HETEROCHROMATIN; FILAMENTOUS GROWTH; MOLECULAR-MODEL; YEAST; HEAD; REPLICATION
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8/9/38 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08449842 Genuine Article#: 286LH Number of References: 44
Title: Identification of a boundary domain adjacent to the potent human cytomegalovirus enhancer that represses transcription of the divergent UL127 promoter
Author(s): Angulo A; Kerry D; Huang H; Borst EM; Razinsky A; Wu J; Hobom U; Messerle M; Ghazal P (REPRINT)
Corporate Source: SCRIPPS CLIN & RES INST,DEPT IMMUNOL & MOL BIOL, DIV VIROL R307B, 10550 N TORREY PINES RD/LA JOLLA//CA/92037 (REPRINT); SCRIPPS CLIN & RES INST,DEPT IMMUNOL & MOL BIOL, DIV VIROL R307B/LA JOLLA//CA/92037; MAX VON PETTENKOFER INST,/MUNICH//GERMANY/
Journal: JOURNAL OF VIROLOGY, 2000, V74, N6 (MAR), P2826-2839
ISSN: 0022-538X Publication date: 20000300
Publisher: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW, WASHINGTON, DC 20005-4171
Language: English Document Type: ARTICLE
Geographic Location: USA; GERMANY
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: VIROLOGY
Abstract: Transcriptional repression within a complex modular promoter may play a key role in determining the action of enhancer elements. In human cytomegalovirus? the major immediate-early promoter (MIEP) locus contains a highly potent and complex modular enhancer. Evidence is presented suggesting that sequences of the MIEP between nucleotide positions -556 and -673 function to prevent transcription activation by enhancer elements from the UL127 open reading frame divergent promoter. Transient transfection assays of reporter plasmids revealed repressor

sequences located between nucleotides -556 and -638. The ability of these sequences to confer repression in the context of an infection was shown using recombinant viruses generated from a bacterial artificial chromosome containing an infectious human cytomegalovirus genome, In addition to repressor sequences between -556 and -638, infection experiments using recombinant virus mutants indicated that sequences between -638 and -673 also contribute to repression of the UL127 promoter. On the basis of in vitro transcription and transient transfection assays, we further show that interposed viral repressor sequences completely inhibit enhancer-mediated activation of not only the homologous but also heterologous promoters. These and other experiments suggest that repression involves an interaction of host-encoded **regulatory** factors with defined promoter sequences that have the property of proximally interfering with upstream enhancer elements in a chromatin-independent manner. Altogether, our findings establish the presence of a boundary domain that efficiently **blocks** enhancer-promoter interactions, thus explaining how the enhancer can work to selectively activate the MIEP.

Identifiers--KeyWord Plus(R): IMMEDIATE-EARLY PROMOTER; **WINGED HELIX PROTEINS**; EARLY GENE; ESCHERICHIA-COLI; TARGET SEQUENCE; CAP SITE; EXPRESSION; IE2; VIRUS; DROSOPHILA

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End of Result Set

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L1: Entry 8 of 8 File: DWPI Mar 2, 2004

DERWENT-ACC-NO: 2001-112567
DERWENT-WEEK: 200417

TITLE: Identifying inhibitors of staphylococcal SarA (accessory regulator) which are useful for treating staphylococcal infections, comprises using specific binding sites of SarA protein on an accessory gene regulator locus

INVENTOR: HURLBURT, B K; RECHTIN, T M ; SMELTZER, M S

PRIORITY-DATA: 1999US-142793P (July 8, 1999), 2000US-0612549 (July 7, 2000)

Search Selected Search ALL Clear

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 6699662 B1	March 2, 2004		000	C12Q001/68
WO 200103686 A2	January 18, 2001	E	079	A61K031/00
AU 200059177 A	January 30, 2001		000	A61K031/00

INT-CL (IPC): A61 K 31/00; C07 H 21/62; C12 P 19/34; C12 Q 1/68

Previous Doc Next Doc Go to Doc#

UniProtKB/TrEMBL entry Q9F0R1

Note: most headings are clickable, even if they don't appear as links. They link to the user manual or other documents.

Entry information

Entry name	**Q9F0R1_STAAU**
Primary accession number	**Q9F0R1**
Secondary accession numbers	None
Entered in TrEMBL in	Release 16, March 2001
Sequence was last modified in	Release 16, March 2001
Annotations were last modified in	Release 26, March 2004

Name and origin of the protein

Protein name	**SarR**
Synonyms	None
Gene name	**Name: sarR**
From	Staphylococcus aureus [TaxID: 1280]
Taxonomy	Bacteria; Firmicutes; Bacillales; Staphylococcus.

References

[1] NUCLEOTIDE SEQUENCE.
STRAIN=RN6390;
Cheung A.L., Manna A.C.;
Submitted (NOV-1999) to the EMBL/GenBank/DDBJ databases.

Comments

None

Cross-references

EMBL	AF207701; AAG35715.1; -; Genomic_DNA.	[EMBL / GenBank / DDBJ] [CoDingSequence]
PIR	B90028; B90028.	
SMR	Q9F0R1; 1-115.	
GO	GO:0003677; Molecular function: DNA binding *(inferred from electronic annotation).* GO:0006355; Biological process: regulation of transcription, DNA-dependent *(inferred from electronic annotation).* GO:0006350; Biological process: transcription *(inferred from electronic annotation).* QuickGo view.	
InterPro	IPR010166; Staph_reg_Sar. IPR011991; Wing_hlx_DNA_bd. Graphical view of domain structure.	
TIGRFAMs	TIGR01889; Staph_reg_Sar; 1.	

ProDom	[Domain structure / List of seq. sharing at least 1 domain]
HOGENOM	[Family / Alignment / Tree]
ProtoMap	Q9F0R1.
PRESAGE	Q9F0R1.
ModBase	Q9F0R1.
SWISS-2DPAGE	Get region on 2D PAGE.
UniRef	View cluster of proteins with at least 50% / 90% / 100% identity.

Keywords

DNA-binding; Transcription; Transcription regulation.

Features

None

Sequence information

Length: **115** **AA** Molecular weight: **13669** **Da** CRC64: **D2CE40E2DB234DBD** [This is a checksum on the sequence]

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        10         20         30         40         50         60
MSKINDINDL VNATFQVKKF FRDTKKKFNL NYEEIYILNH ILRSESNEIS SKEIAKCSEF

        70         80         90        100        110
KPYYLTKALQ KLKDLKLLSK KRSLQDERTV IVYVTDTQKA NIQKLISELE EYIKN
```

Q9F0R1 in FASTA format

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of both extracellular and cell wall virulence determinants, these promoter-gfpUV constructs should be useful to characterize many aspects of S. aureus gene regulation in vivo.

Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Endocarditis, Bacterial--genetics--GE; *Gene Expression Regulation, Bacterial; *Promoter Regions (Genetics); * **Staphylococcal** Infections--genetics--GE; * **Staphylococcus** aureus--genetics--GE; Animals; Genes, Reporter; Luminescent Proteins--biosynthesis--BI; RNA; Rabbits; Recombinant Fusion Proteins--biosynthesis--BI

CAS Registry No.: 0 (Luminescent Proteins); 0 (RNA, recombinant); 0 (Recombinant Fusion Proteins); 63231-63-0 (RNA)

Record Date Created: 19981224

Record Date Completed: 19981224

7/9/3 (Item 3 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

12372435 PMID: 9683479

Transcriptional analysis of different promoters in the sar locus in Staphylococcus aureus.

Manna A C; Bayer M G; Cheung A L

Laboratory of Bacterial Pathogenesis and Immunology, The Rockefeller University, New York, New York 10021, USA.

Journal of bacteriology (UNITED STATES) Aug 1998, 180 (15) p3828-36, ISSN 0021-9193 Journal Code: 2985120R

Contract/Grant No.: AI30061; AI; NIAID; AI37142; AI; NIAID

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Document type: Journal Article

Languages: ENGLISH

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Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

The expression of extracellular virulence determinants in **Staphylococcus** aureus is controlled by a 510-nucleotide RNA molecule (RNAIII) which is a part of the agr system. The agr operon, which encodes a multicomponent signal transduction system, is partially under the influence of an unlinked regulatory locus called **sar** . The **sar** locus is composed of three overlapping transcripts, designated **sarA** (0.56 kb), sarC (0.8 kb), and sarB (1.2 kb), originating from the P1, P3, and **P2** **promoters** , respectively. In this study, we analyzed the differential expression of these promoters by using transcriptional fusion with the xylE reporter gene to study the activation of the **sar** locus. The data confirm the existence of three independent promoters with different promoter activities. Maximal promoter activity was observed with the combined fusion of **P2** -P3-P1 **promoters** . Expression studies with a sigB mutant revealed that the P3 promoter is SigB dependent. Analysis of these transcriptional fusions in a **sarA** mutant and in complemented strains with each of the **sar** transcriptional units revealed that the **sar** locus is autoregulatory, with **SarA** acting as a positive regulator. From various transcriptional fusion studies of the upstream region of the P1 promoter, we have localized a 34-bp sequence which seems to play a role in down-modulating P1 transcription. Using heparin-Sepharose and DNA-specific columns, we partially purified a 12-kDa protein, possibly a repressor, which binds to the promoter regions upstream of P2 and P1 and which also binds to the 34-bp sequence. These data indicated that the regulation of the **sar** locus is complex and may involve the **sar** gene product(s) and other regulatory protein(s).

Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.

Descriptors: *Dioxygenases; *Genes, Bacterial; *Operon; *Promoter Regions (Genetics); * **Staphylococcus** aureus--genetics--GE; *Transcription, Genetic ; Base Sequence; Gene Expression Regulation, Bacterial; Genes, Reporter; Genetic Complementation Test; Molecular Sequence Data; Nucleic Acid Conformation; Oxygenases--biosynthesis--BI; Plasmids; Recombinant Fusion Proteins--biosynthesis--BI; Repetitive Sequences, Nucleic Acid; **Staphylococcus** aureus--pathogenicity--PY; Virulence--genetics--GE

CAS Registry No.: 0 (Plasmids); 0 (Recombinant Fusion Proteins)

Enzyme No.: EC 1.13. (Oxygenases); EC 1.13.11 (Dioxygenases); EC 1.13.11.2 (catechol-2,3-dioxygenase)

Record Date Created: 19980820

Record Date Completed: 19980820

7/9/4 (Item 4 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

12149033 PMID: 9446568

Molecular interactions between two global regulators, sar and agr, in Staphylococcus **aureus.**

Chien Y; Cheung A L

Laboratory of Bacterial Pathogenesis and Immunology, The Rockefeller University, New York, New York 10021, USA.

Journal of biological chemistry (UNITED STATES) Jan 30 1998, 273 (5) p2645-52, ISSN 0021-9258 Journal Code: 2985121R

Contract/Grant No.: AI30061; AI; NIAID; AI37142; AI; NIAID

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Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Subfile: INDEX MEDICUS

The expression of many virulence determinants in **Staphylococcus** aureus is controlled by regulatory loci such as agr and **sar** . We have previously shown that the **SarA** protein is required for optimal transcription of RNAII and RNAIII in the agr locus. To define the specific molecular interaction, we overexpressed **SarA** as a glutathione S-transferase (GST) fusion protein by cloning the 372-base pair (bp) **sarA** gene into the vector. The purified GST- **SarA** as well as cleaved **SarA** were able to bind specifically to the P2, P3, and the combined **P2** -P3 **promoter** fragments of agr in gel shift assays. Using monoclonal antibodies to **SarA** , we found that **SarA** is a part of the retarded protein-DNA complex as evidenced by the formation of a supershifted band. The **SarA** binding site on the agr promoter, mapped by DNase I footprinting assay, covered a 29-bp region between the **P2** and P3 **promoters** devoid of any direct repeats. A synthetic 45-bp fragment encompassing the 29-bp sequence also bound the **SarA** protein in band shift assays. Serial in-frame deletion analysis of **sarA** revealed that, with the exception of 15 residues in the N terminus, almost all of **SarA** (residues 16-124) is essential for agr binding activity. Northern analysis confirmed that only the **sar** mutant clone containing a truncated **sarA** gene with a 15-residue deletion in the N terminus (**SarA16** -124) could activate agr transcription to a level approaching that of the full-length counterpart (**SarA1** -124). Taken together, these data indicated that **SarA** is a DNA-binding protein with binding specificity to the P2 and P3 interpromoter region of agr, thereby activating RNAII and RNAIII transcription.

Tags: Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't,

Search	Most Recent Queries	Time	Result
#8	**Related Articles** for **PubMed** (Select **11159982**) sort by: **PublicationDate**	09:51:16	198
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#5	Search **sarr**	09:21:19	600
#3	Search **marr sarr**	09:15:46	1
#1	Search **marr family**	09:14:16	79

Mol Gen Genet. 1999 Sep;262(2):323-31. Related Articles, Links

Interactive regulatory pathways control virulence determinant production and stability in response to environmental conditions in Staphylococcus aureus.

Lindsay JA, Foster SJ.

Department of Molecular Biology and Biotechnology, University of Sheffield, Western Bank, UK.

The accessory gene regulator (agr) and staphylococcal accessory regulator (sar) loci are important regulators of toxin production in Staphylococcus aureus. In this study we examined how environmental conditions degree of aeration and salt concentration - affect the transcription and translation of mRNAs for alpha-haemolysin (Hla) and serine protease (Ssp) via these pathways and influence the stability of these proteins. Using Northern analysis, we have confirmed earlier observations that sarA is involved in the upregulation of RNAIII, the effector molecule encoded by the agr locus. However, this effect was abolished in highly aerated cultures. While sarA does appear to have an up-regulatory effect on hla transcription that is independent of agr, we propose that the PC1839 (sarA) mutant produces less alpha-haemolysin activity mainly as a result of post-translational inactivation by proteases. The most obvious phenotypic feature of PC1839 (sarA) is the upregulation of proteases. In this study we show that ssp is repressed by SarA at the transcriptional level. Western analysis using an anti-alpha-haemolysin antibody identified a major breakdown product that is only present in the supernatant of strains that are overexpressing serine protease. We have also confirmed that agr exerts a significant regulatory influence on hla at the level of translation, as well as transcription. Finally, the addition of salt upregulates ssp transcription and dramatically downregulates transcription of hla, and is an example of an environmental parameter that affects toxin production independently of agr and sarA. How environmental signals are transduced to control alpha-haemolysin and serine protease production, activity and stability at multiple levels are discussed.

PMID: 10517329 [PubMed - indexed for MEDLINE]

Mol Gen Genet. 1999 Sep;262(2):323-31. Related Articles, Links

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Department of Molecular Biology and Biotechnology, University of Sheffield, Western Bank, UK.

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PMID: 10517329 [PubMed - indexed for MEDLINE]

DOCUMENT-IDENTIFIER: US 20030171563 A1
TITLE: Regulators of bacterial virulence factor expression

Summary of Invention Paragraph:
[0003] Virulence factor regulation. The expression of many virulence factors of S. aureus is controlled by the products of several loci. In laboratory cultures, virulence factor expression is altered as the bacteria transition from the exponential phase to stationary phase of growth (Abbas-Ali and Colman, 1997; Ji et al., 1995; Novick et al., 1990). Throughout the post-exponential phase of growth the components encoded by agr, sae, ar, and at least six MarR-family regulators (sarA and the Sar-homologues) function together to repress the transcription of genes encoding cell-surface virulence factors while increasing the transcription of genes encoding extracellular toxins and enzymes (Janzon and Arvidson, 1990; Novick et al., 1993). Measurements of the levels of translation products in strains with regulatory gene mutations have been complicated by the lack of quantitative, reproducible, specific assays. However, the reduction of exponential phase cell-surface proteins and the increase in all but one of the regulated post-exponential phase proteins that have been examined mirrors the pattern of their transcription (Bjorklind and Arvidson, 1980; Morfeldt et al., 1996; Novick et al, 1993).

Summary of Invention Paragraph:
[0004] A model of virulence factor regulation. The current understanding of the function of components encoded by agr, sae, ar, sarA, and the Sar-homologues, has lead to a model that describes the mechanism of virulence factor regulation. Central in the model is agr (FIG. 1, item 1) (Peng et al, 1988). This locus consists of divergent messages transcribed from adjacent promoters designated P2 and P3 (Janzon and Arvidson, 1990; Kornblum et al., 1990). The P2 promoter message, RNAII, encodes four proteins, AgrA, AgrB, AgrC, and AgrD (Kornblum et al., 1990; Norvick et al., 1995). These proteins are involved in a partially self-inducing, pheromone-sensing, signal transduction circuit. Two of the agr-encoded proteins share sequence homology with components of other bacterial signal transduction systems (Norvick et al., 1995). These proteins, AgrC and AgrA, act as a histidine-kinase sensor and a response regulator, respectively. The activating signal of the agr system is a peptide pheromone modified from the pre-peptide protein AgrD (Ji et al., 1995). AgrB is believed to be the enzyme responsible for the maturation (modification and secretion) of the peptide pheromone (Ji et al., 1997).

Summary of Invention Paragraph:
[0005] The agr system begins to function when AgrC binds the AgrD-derived peptide signal (Ji et al., 1997, Novick et al., 1995). Like other bacterial sensor proteins, the binding of the signal to the sensor protein initiates an autophosphorylation event and presumably, a concomitant activating conformational change to AgrC (Kornblum et al., 1990; Lina et al., 1998). The phosphate group on AgrC is thought to be transferred to the regulator protein, AgrA. This phosphate transfer results in an activating, again presumably conformational, change to AgrA. Unlike other bacterial signal transduction systems where the activated regulator protein directly initiates the transcription of target promoters, genetic evidence provides support that activated AgrA functions with the translation product of a genetically unlinked locus named sarA to up-regulate transcription from the agr promoters (Cheung et al., 1994; Cheung et al., 1995). The agr promoters are the only known targets of AgrA. The sarA product, SarA (FIG. 1, item 2), has been shown to bind DNA between the two agr promoters (Chien et al., 1998; Heinrichs et al., 1996; Rechtin et al., 1999). While the activation of agr has been genetically and biochemically examined, the specific roles of AgrA and SarA remain unclear. Regardless of the mechanism, the result of the increase in P2 and P3 transcription is an amplification of the activating circuit encoded by RNAII and the high-level production of a 514-ribonucleotide RNA known as RNAIII (Janzon and Arvidson, 1990; Morfeldt et al., 1996; Novick et al., 1993).

Summary of Invention Paragraph:

[0006] SarA is transcribed from three different overlapping messages. These messages, from largest to smallest, are known as "B", "C", and "A" (Bayer et al., 1996). All the messages are initiated from distinct upstream promoters (P2, P3, and P1, respectively) and end at a common terminator downstream of the SarA open reading frame. The Pi transcript is the dominant message in S. aureus 8325-4 derived laboratory strains. Transcription from the sarA P1 and P2 promoters is dependent on primary sigma factor in S. aureus (.sigma..sup.A), while the sarA P3 promoter is dependent on the multiple-stress-responsive sigma factor (.sigma..sup.B) (Deora et al., 1997; Palma and Cheung, 2001). Virulence factor regulation has been studied in S. aureus strains derived from 8325-4. These strains have a mutation in the rsbu-encoded phosphatase and phenotypically have a reduced stress response (Kullik et al., 1998). Recent experiments have shown that full .sigma..sup.B activity enhances transcription of sarA, decreases transcription of agr, and modulates virulence factor gene transcription (Chan and Foster, 1998; Kullik et al., 1998).

Summary of Invention Paragraph:
[0007] In addition to encoding SarA, the three sar messages play a direct role in virulence factor regulation. For example, transcriptional attenuation of the gene encoding protein A (spa) requires the sarA "A" message in sar-minus strains, while the sarA "B" and "C" messages complement agr-minus strains (Bayer et al., 1996; Cheung and Projan, 1994). These data need to be viewed with caution. The DNA fragments encoding the sarA messages were cloned on multi-copy number plasmids. These constructs may be expected to alter the concentration of SarA which can have a profound effect on the levels, and therefore activity, of the other regulators involved in the control of virulence factor expression (see below). Furthermore, interpretation of the sarA complementation studies is difficult because dissimilar phenotypes have been reported in sarA-minus strains sharing the same genetic background (Chan and Foster, 1998; Chien et al., 1998).

Summary of Invention Paragraph:
[0008] The MarR-family and the SarA-homologues. Complexity is added to the model of virulence factor regulation by the recent identification of additional regulatory proteins that modulate virulence factor expression. These modulators are members of the MarR-family of bacterial regulators (ExPASy Prosite, PS01117). The MarR-family of regulators is named after a repressor of regulons involved in multiple antibiotic resistance and oxidative stress in E. coli (Cohen et al., 1993). This family also includes regulators of anabolic pathways, toxins, sporulation, and protease production (Ludwig et al., 1995; Ruppen et al., 1988; Thomson et al., 1997). The majority of MarR homologues are repressors; however, at least one family member appears to act as an activator (Thomson et al., 1997). Thirteen MarR-family members can be identified by BLASTP searches of the S. aureus (FIG. 11). The genes encoding these proteins are present in the genome databases of strains N315, Mu50, COL, and 8325-4 (Kuroda et al. (2001)). Included in this group are SarA, SarR, SarS, SarT, SarU, and Rot. With regard to the regulation of toxin production, the remaining homologues have yet to be characterized in regard to their effect on toxin production. This includes TcaR (B. Berger-Bachi, personal communication). Even though the genes encoding these Sar-homologues are distributed non-uniformly around the genome, an operon naming system is utilized (Kuroda et al. (2001)).

Summary of Invention Paragraph:
[0009] Rot, SarR, SarS, SarT, and SarU are all components of the network of regulators that includes RNAIII and SarA. Genetic and biochemical data strongly implicate Rot and SarS (FIG. 1, item 3) in the repression of toxin gene transcription and the activation of cell-surface gene transcription. While we identified rot by screening a pool of transposon mutants, SarS was isolated from lysates using target promoter DNA fragments linked to magnetic beads (McNamara et al., 2000; Tegmark et al., 2000). In vitro, SarS has been shown to bind the promoter region of hla (the gene encoding the .alpha.-toxin), sspA (the gene encoding the V8 protease), spa, and rnaIII (the gene encoding agr RNAIII), although an effect on transcription was only seen with hla and spa (Cheung et al., 2001; Tegmark et al., 2000). SarR

(FIG. 1, item 4) was isolated using a DNA-column with sarA P2 promoter fragments (Marrack and Kappler, 1990). When compared to wild-type strains, sarR mutant strains show increased expression of SarA. SarT (FIG. 1, item 6) was identified in the sequence of the S. aureus strain COL genome by homology to SarA (Schmidt et al., 2001). SarT is required for the transcriptional repression of rnaIII and hla. Although not included in our model, as we described with rot, an element encoded by agr appears to down-regulate expression of SarT (McNamara et al., 2000; Schmidt et al., 2001). Transcription of hla, the only virulence factor gene examined to date in sarT-minus strains, is dependent upon the repression of SarT by SarA (Schmidt et al., 2001). SarU (FIG. 1, item 5) appears to be required for transcription of both agr and sarA.

Summary of Invention Paragraph:
[0010] It is clear that the Sar-homologues affect the transcription of other Sar-homologues genes as well as agr. As mentioned above, the levels of SarS and SarT are dependent on the level of SarA (Cheung et al., 2001; Schmidt et al., 2001) and the level of SarA is related to the level of SarR (Manna and Cheung, 2001). Preliminary data indicate that Rot is involved in the down-regulation of transcription of SarS. Therefore, increased expression of a Sar-homologue can influence the level of other Sar-homologues, regulators, and ultimately, virulence factor genes. As mentioned above, sarA when cloned on a multi-copy plasmid and moderately overexpressed in S. aureus both negatively affects bacterial growth and reduces transcription of genes that are normally positively regulated by SarA in wild-type strains (Tegmark et al., 2001).

Summary of Invention Paragraph:
[0011] DNA binding sites and the structure of the Sar homoloques. The DNA recognition sequence for recombinant SarA (rSarA) binding has been examined by several groups. DNase I footprinting and sequence analysis of sequences upstream of regulated genes defined specific "AT"-rich binding sites and have shown that rSarA binds as a dimer protecting between 20 to 38-bp of DNA depending on the report (Cheung and Projan, 1994; Rechtin et al., 1999; Tegmark et al., 2001). In one study, rSarA was shown to bind linear DNA with limited sequence specificity (Tegmark et al., 2001). Instead, DNA fragments with a minimum "AT" content of 76% were shown to be sufficient for rSarA-binding. In this same study, sequences with slightly higher binding affinities were also found. These sequences corresponded to the specific sequences that were reported by the other investigators (Tegmark et al., 2001). It is difficult to believe that SarA is a nonspecific DNA binding protein. As the archetype for the Sar-homologues, the data of Tegmark et al. would imply that all the SarA-homologues are nonspecific DNA binding proteins (Tegmark et al., 2001). This leads to the question of how mutations in different Sar-homologue genes confer different phenotypes. While the levels of the various Sar-homologues may play a role, SarA and the Sar-homologues may require supercoiled rather than linear DNA for specific binding.

Summary of Invention Paragraph:
[0012] Crystal structures were determined for a rSarA monomer and a monomeric rSarA-6mer DNA complex (Schumacher et al., 2001a). rSarA has four .alpha.-helices domains and two inducible regions that consist of a .beta.-hairpin and a carboxy-terminal loop. Studies of the rSarA-DNA complex revealed that the inducible domains in rSarA undergo extensive conformational changes that result in the formation of extended .alpha.-helices which wrap around DNA having a D-DNA-like conformation. Caution is indicated in accepting these data because they were obtained for a monomeric form of rSarA bound to a short DNA sequence (Schumacher et al., 2001b). DNase I protection and gel shift assays demonstrate that SarA binds DNA as a dimer, protects at least 20-bp of DNA, and introduces bends into the target DNA (Rechtin et al., 1999; Tegmark et al., 2001).

Summary of Invention Paragraph:
[0013] Structural and binding properties have also been determined for recombinant SarR (rSarR).

DNase I protection and gel shift assays have demonstrated that rSarR binds DNA surrounding all three sarA promoters, although a specific DNA binding sequences were not defined (Liu et al., 2001; Manna and Chueng, 2001). Like rSarA, rSarR was shown to bind to DNA as a dimer. The crystal structure studies revealed that rSarR has both a classic helix-turn-helix motif for DNA binding in the major groove and a loop region involved in recognition of the minor groove (Liu et al., 2001). rSarR was shown to interact with approximately 27 bp of target DNA and to induce bends within the target DNA (Liu et al., 2001; Manna and Chueng, 2001). It is reasonable to assume that the characterized Sar-homologues that are most closely related to SarR (SarA, Rot, and SarT) bind to DNA as dimers using the helix-turn-helix motif and loop region and act as DNA-bending proteins. It is unknown if SarR, Rot, and SarT can form heterodimers. In contrast, SarS and SarU have two DNA-binding domains and probably bind DNA as a monomer, although other higher ordered quaternary structures are possible.

Summary of Invention Paragraph:
[0015] ArlRS encodes a two component signal transduction system that has been shown to affect virulence factor gene transcription (FIG. 1, item 8) (Fournier et al., 2001). Mutations in arlRS increase the transcription of hla, hlb, ssp, and spa. The observed up-regulation of gene transcription in the mutant strain is reflected in the secreted products. Analysis of mutant strains showed that an ArlSR mutation increases synthesis of agr RNAII and RNAIII and decreases the synthesis of SarA.

Brief Description of Drawings Paragraph:
[0055] FIG. 3. BLASTP alignment of the rot gene product (Rot) and the S. aureus regulatory protein SarA, (e value=5.9) and the S. epidermidis SarA (e value=0.49). Rot was used as the query sequence against the non-redundant database at the National Center for Biotechnology Information. Identities are shown, (+) denotes similarity, and numbers at right refer to amino acids in the respective proteins.

Brief Description of Drawings Paragraph:
[0058] FIG. 6. BLASTP alignment of the gene products of rlp and sarA. Identities are shown, (+) denotes similarity, and numbers at right refer to amino acids in the respective proteins.

Detail Description Paragraph:
[0249] FIG. 1. A Model for the regulation of staphylococcal virulence factors. A single S. aureus is delineated by the large hatched circle. Genes are represented by boxes and promoters are labeled "P". With the exception of RNAIII (arrow and ladder-like structure) and the .alpha.-toxin message (arrow), mRNA is depicted using straight lines with triangular arrowheads. 1. The P2 and P3 operons of the agr locus. The agr P2 promoter transcribes RNAII, which encodes AgrA, AgrC, AgrD, and AgrB. The agr P3 promoter transcribes RNAIII, which encodes 6-toxin (hld). AgrC is activated by autophosphorylation after binding the AgrD-derived peptide pheromone. The phosphate group (circle) is transferred from AgrC to AgrA. Activated AgrA functions to increase transcription RNAII and RNAIII. RNAIII is associated with the down-regulation of cell-surface protein genes and the up-regulation of extracellular protein genes including .alpha.-toxin (hla). RNAIII is required for translational control of the hla message. Translation of .delta.-toxin (short lines) is the only known protein product of RNAIII. 2. The sar locus is transcribed on three messages each encoding SarA. SarA is required for the activation of agr P2 promoter. SarA, the sar transcripts, or an uncharacterized protein encoded on the SarA messages is involved in the down-regulation of transcription of the gene encoding protein A (spa). 3. Rot and SarS are involved in the up-regulation of cell-surface protein gene transcription and the down-regulation of extracellular protein gene transcription. Rot represses transcription of sarS. 4. SarR represses transcription of sarA. 5. SarU is required for of sarA and agr transcription. 6. SarT is a repressor of agr and hla. 7. The sae locus encodes a phosphate-transferring two-component signal transduction system that affects both cell surface and extracellular protein expression by an unknown mechanism. 8. The arlRS locus encodes a phosphate-transferring two-component signal transduction system that down-regulates transcription of spa and hla. Not shown in this model are affects of components of arlRS that

up-regulate transcription from the agr P2 and P3 promoters and down-regulate transcription on sarA. 9. The sarA P3 and a second sarS promoter are dependent upon .sigma..sup.B. Rot down-regulates sB expression.

Detail Description Paragraph:
[0251] FIG. 3. BLASTP alignment of the rot gene product (Rot) and the S. aureus regulatory protein SarA, (e value 5.9) and the S. epidermidis SarA (e value=0.49). Rot was used as the query sequence against the non-redundant database at the National Center for Biotechnology Information. Identities are shown, (+) denotes similarity, and numbers at right refer to amino acids in the respective proteins.

Detail Description Paragraph:
[0254] FIG. 6. BLASTP alignment of the gene products of rlp and sarA. Identities are shown, (+) denotes similarity, and numbers at right refer to amino acids in the respective proteins.

Detail Description Paragraph:
[0292] A BLASTP search using a conceptional translation of the predicted 161 amino acid protein identified hypothetical proteins (GenBank U89914 and Swiss-protein P54182) and a region of homology to SarA from S. aureus and S. epidermidis (FIG. 3). The transposon inactivated gene was named rot (repressor of toxins) because loss of a wild type allele results in the restoration of protease and .alpha.-toxin activities to S. aureus PM466 and to reflect the fact that it has homology to known transcriptional regulators and acts as a repressor of toxin synthesis.

Detail Description Paragraph:
[0304] This example describes an erythromycin insertional knockout mutant of a new regulator in S. aureus, and the effect of the knockout on coagulase and .alpha.-toxin activities and messages. Additionally, it describes the examination of total extracellular protease activity and the effect of the mutation on the levels of RNAIII and the SarA messages.

Detail Description Paragraph:
[0306] BLASTP searches of the partially completed DNA sequence from S. aureus COL and GenBank genome, using the predicted rot gene product of Example 1, identified the staphylococcal regulator SarA, as well as previously uncharacterized gene encoding a potential 247 amino acid protein named herein rlp for rot-like protein (GenBank accession number AF288788). Rlp and SarA share approximately 34% identity and 56% homology over the range of reported amino acids (FIG. 6). The rlp locus was amplified by PCR and cloned to create plasmid pJM730 (Table 4).

Detail Description Paragraph:
[0315] Total RNA from strains RN6390, PM734, PM743, and PM466 isolated during either exponential and post-exponential phase of growth was analyzed for, .alpha.-toxin, protein A, rnaii, rnaiii, and SarA messages by primer extension. The results from the exponential phase cultures are shown in FIGS. 9A, 9B, and 9C.

Detail Description Paragraph:
[0320] Levels of coagulase and .alpha.-toxin messages mirrored the activity data providing evidence that the rlp gene product affects the transcription of virulence factor genes. A comparison of RNAIII levels in strains RN6390 (wild-type) and PM734 (rlp::erm) was made to distinguish between an agr-dependent or independent regulation. Unlike in RN6390, RNAIII could not be detected in RNA isolated from post-exponential phase cultures of PM734. These data provide supporting evidence that the rlp, like sar, encodes an activator of agr or is required for the transcription of sarA. To distinguish between these possibilities, the levels of sarA messages in RN6390 and PM734 were compared and found to be identical, providing support that the rlp gene products acts directly upon the agr promoters and that the

phenotypic effect of the rlp::erm mutation is mediated by RNAIII.

Detail Description Paragraph:
[0332] Total cellular RNA was isolated from 3 and 10 hour cultures (OD.sub.540 of approximately 0.2 and 3.0, respectively) of S. aureus and purified, as described by McNamara et al. (2000). Gene-specific primers for the genes encoding protein A (spa), .alpha.-toxin (hla), RNAII, and RNAIII were 5'-CCTAAAGTTACAGATGCAATACC-3' (SEQ ID NO:17), 5'-CGAGGGTTAGTCAAAGTTG-3' (SEQ ID NO:18), and 5'-GTGCCATTGAAATCACTCCTT-3' (SEQ ID NO:19), respectively. The SarA primers have been previously described (Bayer et al, 1996). Primers were end-labeled with .gamma.-.sup.32P ATP using T4 polynucleotide kinase (Promega BioTech, Madison, Wis.) as described by the manufacturer. Complementary DNA was synthesized using 200 U Superscript II (Gibco BRL, Grand Island, N.Y.) in reactions with 1.times. First Strand Buffer, 0.01 M DTT, 0.05 .mu.g/.mu.l Actinomycin D, 0.1 mM dNTPs. The concentration of total RNA in reactions with hla-, RNAII-, and RNAIII-specific primers was 5 ug/ml. A total of 15 ug/ml total RNA was used with the sar- and spa-specific primers. Reaction mixtures were incubated at 50 C. for 50 min. DNA sequence was determined for the promoter regions of the genes encoding protein A, .alpha.-toxin, RNAII, and RNAIII, using Thermo Sequenase (USE Corporation, Cleveland, Ohio) chemistry on pJM764, pJM765, and pJM440, respectively. Due to the intensity of the signals derived from the RNAIII primer extension products, these samples were diluted 1:15 prior to loading on the gel. Samples were subjected to electrophoresis through 6% polyacrylamide gels in Glycerol Tolerant Buffer (0.1 M Tris Base, 28 mM taurine, 0.5 mM Na.sub.2EDTA. Intensities of bands were determined from scanned gels using a NIH Image.

Detail Description Paragraph:
[0349] Chan, P. F. and S. J. Foster. Role of SarA in virulence determinant production and environmental signal transduction in Staphylococcus aureus. J. Bacteriol. 180: 6232-6241, 1998.

Detail Description Paragraph:
[0351] Cheung, A. L., Schmidt, K., Bateman, B. and A. C. Manna. SarS, a SarA homolog repressible by agr, is an activator of protein A synthesis in Staphylococcus aureus. Infect Immun 69:2448-2455, 2001.

Detail Description Paragraph:
[0352] Cheung, A. L. and S. J. Projan. Cloning and sequencing of sarA of Staphylococcus aureus, a gene required for the expression of agr. J. Bacteriol. 176:4168-4172, 1994.

Detail Description Paragraph:
[0355] Cheung, A. L. and S. J. Projan. Cloning and sequencing of sarA of Staphylococcus aureus, a gene required for the expression of agr. J. Bacteriol. 176:4168-4172, 1994.

Detail Description Paragraph:
[0357] Chien, Y., Manna, A. C. and A. L. Cheung. SarA level is a determinant of agr activation in Staphylococcus aureus. Molec. Microbiol. 39:991-1001, 1998.

Detail Description Paragraph:
[0358] Chien, Y., Manna, A. C., Porjan, S. J. and A. L. Cheung. SarA, a global regulator of virulence determinants in Staphylococcus aureus, binds to a conserved motif essential for sar-dependent gene regulation. J. Biol. Chem. 24: 37169-37176, 1999.

Detail Description Paragraph:
[0393] Liu, Y., Manna, A., Li, R., Martin, W. E., Murphy, R. C., Cheung, A. L. and G. Zhang. Crystal structure of the SarR protein from Staphylococcus aureus. Proc. Natl. Acad. Sci. U.S.A. 98:6877-6882, 2001.

Detail Description Paragraph:
[0395] Manna, A. and A. L. Cheung. Characterization of sarR, a modulator of sar expression in Staphylococcus aureus. Infect. Immun. 69:885-889, 2001.

Detail Description Paragraph:
[0409] Rechtin T. M., Gillaspy A. F., Schumacher M. A., Brennan R. G., Smeltzer M. S. and B. K. Hurlburt. Characterization of the SarA virulence gene regulator of Staphylococcus aureus. Molec. Mcrobiol. 33:307-316, 1999.

Detail Description Paragraph:
[0416] Schumacher, M. A., Hurlburt, B. K. and R. G. Brennan. Crystal structures of SarA, a pleiotropic regulator of virulence genes in S. aureus. Nature 409:215-219, 2001a.

Detail Description Paragraph:
[0417] Schumacher, M. A., Hurlburt, B. K. and R. G. Brennan. Correction: Crystal structures of SarA, a pleiotropic regulator of virulence genes in S. aureus. Nature 409:215-219, 2001b.

Detail Description Paragraph:
[0422] Tegmark, K., Morfeldt, E. and S. Arvidson. The virulence gene regulator, SarA appears to be a non-specific DNA binding protein. Molec. Microbiol., in press, 2002.

Previous Doc Next Doc Go to Doc#

embryonic stem cell enhancer sequence. Co-transfection experiments revealed that Genesis is a transcriptional repressor. Genesis mapped to mouse chromosome 4 in a region syntenic with human chromosome 1p31, a site of nonrandom abnormalities in germ cell neoplasia, neuroblastoma, and acute lymphoblastic leukemia. Genesis is a candidate for **regulating** the phenotype of normal or malignant embryonic stem cells.

REGISTRY NUMBERS: 173486-11-8: U41047
DESCRIPTORS:
MAJOR CONCEPTS: Biochemistry and Molecular Biophysics; Development; Genetics; Oncology--Human Medicine, Medical Sciences
BIOSYSTEMATIC NAMES: Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia
ORGANISMS: human (Hominidae); mouse (Muridae)
COMMON TAXONOMIC TERMS: Humans; Primates; Animals; Chordates; Mammals; Nonhuman Vertebrates; Nonhuman Mammals; Rodents; Vertebrates
MOLECULAR SEQUENCE DATABANK NUMBER: amino acid sequence; molecular sequence data; nucleotide sequence; U41047--Genbank
MISCELLANEOUS TERMS: CHROMOSOME 1P31; CHROMOSOME 4; DNA BINDING DOMAIN; EMBRYONAL CARCINOMA CELL; EMBRYONIC STEM CELL; GENE EXPRESSION; GENESIS ; MOLECULAR GENETICS; TUMOR BIOLOGY; **WINGED HELIX TRANSCRIPTIONAL REPRESSOR**
CONCEPT CODES:
03506 Genetics - Animal
03508 Genetics - Human
10060 Biochemistry studies - General
24002 Neoplasms - General
25502 Development and Embryology - General and descriptive
BIOSYSTEMATIC CODES:
86215 Hominidae
86375 Muridae

8/9/35 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08870216 Genuine Article#: 338WV Number of References: 38
Title: The forkhead protein Fkh2 is a component of the yeast cell cycle transcription factor SFF
Author(s): Pic A; Lim FL; Ross SJ; Veal EA; Johnson AL; Sultan MRA; West AG ; Johnston LH; Sharrocks AD; Morgan BA (REPRINT)
Corporate Source: UNIV NEWCASTLE UPON TYNE,SCH MED, SCH BIOCHEM & GENET/NEWCASTLE UPON TYNE NE2 4HH/TYNE & WEAR/ENGLAND/ (REPRINT); UNIV NEWCASTLE UPON TYNE,SCH MED, SCH BIOCHEM & GENET/NEWCASTLE UPON TYNE NE2 4HH/TYNE & WEAR/ENGLAND/; UNIV MANCHESTER,SCH BIOL SCI/MANCHESTER M13 9PT/LANCS/ENGLAND/; NATL INST MED RES,DIV YEAST GENET/LONDON NW7 1AA//ENGLAND/
Journal: EMBO JOURNAL, 2000, V19, N14 (JUL 17), P3750-3761
ISSN: 0261-4189 Publication date: 20000717
Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND
Language: English Document Type: ARTICLE
Geographic Location: ENGLAND
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY; CELL BIOLOGY
Abstract: In the yeast Saccharomyces cerevisiae, the MADS-box protein Mcm1, which is highly related to mammalian SRF (serum response factor), forms a ternary complex with SFF (Swi five factor) to **regulate** the cell cycle expression of genes such as SW15, CLB2 and ACE2, Here we show that the forkhead protein Fkh2 is a component of SFF and is essential for ternary complex formation on the SWI5 and ACE2 promoters. Fkh2 is

essential for the correct cell cycle periodicity of SWI5 and CLB2 gene expression and is phosphorylated with a timing that is consistent with a role in this expression. Furthermore, investigation of the relationship between Fkh2 and a related forkhead protein Fkh1 demonstrates that these proteins act in overlapping pathways to **regulate** cell morphology and cell separation. This is the first example of a eukaryotic transcription factor complex containing both a MADS-box and a forkhead protein, and it has important implications for the **regulation** of mammalian gene expression.

Descriptors--Author Keywords: cell cycle **regulation** ; forkhead ; SFF ; transcription ; yeast

Identifiers--KeyWord Plus(R): **WINGED HELIX PROTEINS**; SACCHAROMYCES-CEREVISIAE; **REGULATED** TRANSCRIPTION; SIGNAL-TRANSDUCTION; BUDDING YEAST; GENE; HEAD; MCM1; EXPRESSION; FAMILY

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8/9/36 (Item 2 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

08784456 Genuine Article#: 328HJ Number of References: 58

Transcriptional regulation of the Staphylococcus aureus collagen adhesin gene (cna) by the staphylococcal accessory regulator (sar). submitted to Molecular Microbiolgy. Boffa, L. C., et al. (1997) Oncology Research 9, 41-51.

Title: A single amino acid substitution in the C terminus of OmpR alters DNA recognition and phosphorylation

Author(s): Tran VK; Oropeza R; Kenney LJ (REPRINT)
Corporate Source: L 220 OREGON HLTH SCI UNIV,DEPT MOL MICROBIOL & IMMUNOL, 3181 SW SAM JACKSON PK RD/PORTLAND//OR/97201 (REPRINT); L 220 OREGON HLTH SCI UNIV,DEPT MOL MICROBIOL & IMMUNOL/PORTLAND//OR/97201

Journal: JOURNAL OF MOLECULAR BIOLOGY, 2000, V299, N5 (JUN 23), P1257-1270
ISSN: 0022-2836 Publication date: 20000623
Publisher: ACADEMIC PRESS LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND
Language: English Document Type: ARTICLE
Geographic Location: USA
Subfile: CC LIFE--Current Contents, Life Sciences
Journal Subject Category: BIOCHEMISTRY & MOLECULAR BIOLOGY

Abstract: In bacteria and lower eukaryotes, adaptation to changes in the environment is often mediated by two-component **regulatory** systems. Such systems provide the basis for chemotaxis, nitrogen and phosphate **regulation** and adaptation to osmotic stress, for example. in Escherichia coli, the sensor kinase EnvZ detects a change in the osmotic environment and phosphorylates the response **regulator** OmpR. Phospho-OmpR binds to the **regulatory** regions of the porin genes ompF and ompC, and alters their expression. Recent evidence suggests that OmpR functions as a global **regulator** , **regulating** additional genes besides the porin genes. Ln this study, we have characterized a previously isolated OmpR2 mutant (V203M) that constitutively activates ompF and fails to express ompC. Because the substitution was located in the C-terminal DNA-binding domain, it had been assumed that the substitution would not affect phosphorylation of the N-terminal domain of OmpR. Our results indicate that this substitution completely eliminates phosphorylation by a small phosphate donor, acetyl phosphate, but not phosphorylation by the kinase EnvZ. The mutant OmpR has altered dephosphorylation kinetics and altered binding affinities to both ompF and ompC sites compared to the wild-type. Thus, a single amino acid substitution in the C-terminal DNA-binding domain has dramatic effects on the N-terminal phosphorylation domain. Most strikingly, we have identified a single base change in the OmpR binding site of ompC that restores high-affinity binding activity by the mutant. We interpret our results in the context of a model for porin gene expression. (C) 2000 Academic Press.

Descriptors--Author Keywords: osmoregulation ; signal transduction ; **winged helix-turn-helix** ; response **regulator** ; two-component **regulatory** system
Identifiers--KeyWord Plus(R): ESCHERICHIA-COLI K-12; POSITIVE **REGULATOR** OMPR; BINDING DOMAIN; TRANSCRIPTIONAL ACTIVATION; SALMONELLA-TYPHIMURIUM; SIGNAL-TRANSDUCTION; MOLECULAR ANALYSIS; CRYSTAL-STRUCTURE; GENE ACTIVATION; PROTEIN

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RBL_SARFL (**P28451**)
Ribulose bisphosphate carboxylase large chain (EC 4.1.1.39) (RuBisCO large subunit) (Fragment). {GENE: Name=rbcL} - Sarracenia flava (Pitcherplant)

Search in UniProtKB/TrEMBL: There are matches to 19 out of 2212675 entries

O98864_SARPR
Maturase (Fragment) {GENE:Name=matK} - Sarracenia purpurea (Pitcher plant) [Plastid; Chloroplast]
Q4L8F0_STAHJ
SarR protein {GENE:Name=sarR; OrderedLocusNames=SH0766} - Staphylococcus haemolyticus (strain JCSC1435)
Q5HDR3_STAAC
Staphylococcal accessory regulator R {GENE:Name=sarR; OrderedLocusNames=SACOL2287} - Staphylococcus aureus (strain COL)
Q5HLV6_STAEQ
Staphylococcal accessory regulator R {GENE:Name=sarR; OrderedLocusNames=SERP1876} - Staphylococcus epidermidis (strain ATCC 35984 / RP62A)
Q5SEQ7_9PERC
Histone H3 (Fragment) - Sarritor frenatus
Q6E6L8_SARPR
5-enol-pyruvylshikimate-phosphate synthase (Fragment) {GENE:Name=EPSPS} - Sarracenia

purpurea (Pitcher plant)

Q6GED9_STAAR
Staphylococcal accessory regulator A homologue {GENE:Name=sarR; OrderedLocusNames=SAR2379} - Staphylococcus aureus (strain MRSA252)

Q7A064_STAAW
SarR protein {GENE:Name=sarR; OrderedLocusNames=MW2213} - Staphylococcus aureus (strain MW2)

Q7A2M3_STAAM
Staphylococcal accessory regulator A homolog {GENE:Name=sarR; OrderedLocusNames=SAV2295} - Staphylococcus aureus (strain Mu50 / ATCC 700699)

Q7A425_STAAN
SarR protein {GENE:Name=sarR; OrderedLocusNames=SA2089} - Staphylococcus aureus (strain N315)

Q8CNC4_STAEP
SarR protein {GENE:OrderedLocusNames=SE1868} - Staphylococcus epidermidis

Q8M8Y7_SARPR
ATP synthase epsilon subunit (Fragment) {GENE:Name=atpE} - Sarracenia purpurea (Pitcher plant) [Plastid; Chloroplast]

Q8M8Y8_SARPR
Maturase (Fragment) {GENE:Name=matK} - Sarracenia purpurea (Pitcher plant) [Plastid; Chloroplast]

Q8SI79_SARFL
Maturase (Fragment) {GENE:Name=matR} - Sarracenia flava (Pitcherplant) [Mitochondrion]

Q8SID1_SARFL
ATP synthase alpha subunit (Fragment) {GENE:Name=atp1} - Sarracenia flava (Pitcherplant) [Mitochondrion]

Q8SMI9_SARPR
Maturase (Fragment) {GENE:Name=matK} - Sarracenia purpurea (Pitcher plant) [Plastid; Chloroplast]

Q9F0R1_STAAU
SarR {GENE:Name=sarR} - Staphylococcus aureus

Q9MTY3_SARFL
ATP synthase beta subunit (Fragment) {GENE:Name=atpB} - Sarracenia flava (Pitcherplant) [Plastid; Chloroplast]

Q9SC18_SARPR
NADH dehydrogenase subunit F (Fragment) {GENE:Name=ndhF} - Sarracenia purpurea (Pitcher plant)

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heterodimer

<biochemistry> A dimer in which the two subunits are different.

One of the best known examples is tubulin that is found as an _ tubulin/_ tubulin dimer. Heterodimers are relatively common and it may be that the arrangement has the advantage that, for example: several different binding subunits may interact with a conserved signalling subunit.

(18 Nov 1997)

Previous: heterodactyl, heterodactylae, heterodactylous, heterodermic, heterodetic peptide
Next: heterodisperse, heterodisulfide reductase, heterodont